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of the
American Veterinary Medical Association

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THE policy of safeguarding the practice of veterinary medicine against unwise intervention was forcefully validated when recent observations in the field of medical research placed more importance than formerly on the transmission of swine erysipelas to human beings.

PUBLIC health, national wealth, and the profits of farming are involved.

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Vol. LXXXIII, N. S. Vol. 36 JULY, 1933 No. 1

IMPROVEMENT

The improved conditions throughout the country generally were reflected in the A. V. M. A. office during the months of May and June in a number of ways. Letters from members in different parts of the country, practically without exception, were more cheerful. Rising prices for live stock and grains were putting farmers in a better mood. With our banking difficulties apparently over and unemployment being reduced, city workers were showing brighter faces. Veterinarians along with others were feeling the effects of the new order of things. As a tangible indication of the improvement, the receipts for dues during May and June were practically at the same level as for the corresponding months of 1929. Quite a number of members who had fallen behind with their dues for 1931 and 1932 paid up, either in part or in full, and several members have already paid up for 1934. Comment on the increase in the number of applications is made on another page. Inquiries concerning reinstatement have been received from quite a few former members, located in the Chicago territory, who want to be "regulars" by the time of the convention. Many of the letters being received from members contain inquiries about the convention, indicating that they are at least thinking about being in Chicago in August. Encouraging signs, all of them.

PROGRAM FOR THE CHICAGO MEETING

Through the splendid coöperation of the officers of the six sections of the A. V. M. A., it is possible to present, over a month before the meeting, a practically complete list of the fifty or more papers which will be presented at the 70th annual convention, as well as an outline of the program for the clinic. It should be understood that there may be changes in the program between now and the meeting. In other words, as presented in this issue of the JOURNAL, the program should be considered as only tentatively arranged.

Negotiations are now being made to group the papers on Bang's disease, and, instead of having this subject presented in no less than three different sections at three different times, plans are under way to arrange a symposium on Bang's disease for one of the general sessions. This arrangement will make it possible for everybody to attend and hear these papers without missing any other part of the program. Bang's disease is a subject that is of such vital interest to practically every veterinarian, whether engaged in practice, sanitary control work, teaching or research, that none will care to miss the papers on this subject.

The statement relative to the tentative arrangement of the papers applies to the program for the clinic as well. Although many of the important features have been decided upon, many of the details will have to be worked out during the next thirty days. A number of the demonstrations which have been arranged for the clinic on Friday will reflect developments in veterinary science brought out by the papers scheduled for presentation before the various sections on Wednesday and Thursday.

An official program will be available when the members register at the Palmer House on August 14. In the official program, all papers and clinical demonstrations will be numbered, thereby enabling the members to follow the program more closely. In addition to the papers scheduled for the sectional meetings, a number of other very timely contributions have been promised and these will be presented at the general sessions scheduled for the afternoon program each day during the week.

As usual, a program of entertainment for the ladies is being arranged, although this will not be so elaborate as in some former years. Plenty of time is being left open, so that the visitors can attend A Century of Progress or see the city. The only evening event definitely scheduled is the banquet, Wednesday

evening, August 16. The President's reception and dance will follow the banquet.

The tentative program follows:

Section on General Practice

Bovine Sterility (Illustrated)—Dr. W. L. Boyd, University of Minnesota, St. Paul, Minn.

*Blood-Testing of Cattle—Dr. R. R. Birch, New York State Veterinary College, Ithaca, N. Y.

Equine Surgery—Dr. Wm. J. Lee, University of Pennsylvania, Philadelphia, Pa.

Large-Animal Practice in the South—Dr. Hamlet Moore, New Orleans, La.

Endometritis of Cows—Dr. Otto Stader, Geneva, Ill.

Developments Concerning Hog Cholera—Dr. C. H. Stange, Iowa State College, Ames, Iowa.

A Century of Hog Cholera—Dr. A. T. Kinsley, Kinsley Laboratories, Kansas City, Mo.

Recognition of the Most Important Infectious Diseases of Swine in the United States—Dr. Frank Breed, Norden Laboratories, Lincoln, Nebr.

Common Ailments of Sheep—Dr. F. E. Stiles, Battle Creek, Mich.

Measuring Fertility in the Ram—Prof. Fred F. McKenzie and Ralph W. Phillips, University of Missouri, Columbia, Mo.

Gas Edema—Dr. C. J. Marshall, University of Pennsylvania, Philadelphia, Pa.

Hemorrhagic Septicemia—Dr. A. F. Schalk, Ohio State University, Columbus, Ohio.

Section on Sanitary Science and Food Hygiene

Canine Rabies Experimental Vaccination (Second and Third Reports)—Drs. M. F. Barnes, A. N. Metcalfe and W. E. Martindale, Bureau of Animal Industry, Department of Agriculture, Harrisburg, Pa., and Dr. W. J. Lentz, University of Pennsylvania, Philadelphia, Pa.

Rabies Vaccine Protection Tests—Drs. John Reichel and J. E. Schneider, Mulford Biological Laboratories, Sharp & Dohme, Glenolden, Pa.

Intradermal Vaccination or the Skin as an Immunological Organ—Dr. C. E. Salsbery, Jensen-Salsbery Laboratories, Kansas City, Mo.

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*Will be transferred to a general session.

New Methods of Immunization in the Control of Anthrax—Dr. Adolph Eichhorn, Lederle Laboratories, Inc., Pearl River, N. Y.

Some Parasites of Oregon Wild Life—Dr. J. N. Shaw, Oregon State Agricultural College, Corvallis, Ore.

*A Practical Interpretation of the Scientific Evidence on the Controversial Points in the Control of Bang's Disease—Drs. C. P. Fitch and C. R. Donham, University of Minnesota, Saint Paul, Minn.

Section on Small Animals

Gastrotomy, Enterotomy and Cystotomy, and Their Indications in the Dog and Cat—Dr. Chas. W. Bower, Topeka, Kan.

Some Observations Relative to Ailments of Inmates in a Zoölogical Collection—Dr. J. A. Campbell, Toronto, Ont.

The Business Side of Our Profession—Dr. J. C. Flynn, Kansas City, Mo.

Practical Anatomical and Physiological Considerations of the Dog—Drs. E. J. Frick and W. M. McLeod, Kansas State College, Manhattan, Kan.

Further Studies in the Control and Hospitalization of Canine Distemper—Dr. M. L. Morris, New Brunswick, N. J.

Blood Chemistry Findings and Their Interpretations in Diseases of Animals—Drs. J. L. Bollman and C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.

Allergic Reactions in the Dog—Dr. P. W. Burns, Texas Agricultural and Mechanical College, College Station, Texas.

Open Reduction and Mechanical Devices in Treating Fractures in Small Animals—Dr. Richard A. Self, Dallas, Texas.

Skin-Grafting in Small Animals—Dr. Richard A. Self, Dallas, Texas.

Section on Research

The Morphology, Culture, Isolation and Immunity Studies of Actinomyces Necrophorus in Calf Diphtheria — Drs. L. H. Scrivner and A. M. Lee, University of Wyoming, Laramie, Wyo.

Studies on Canine Distemper: IV. Immunization of Dogs by Means of Bacterial Products—Dr. A. S. Schlingman, Parke, Davis & Company, Detroit, Mich.

Some Biological Characteristics of the Filtrable Virus of Canine Distemper—Dr. N. J. Pyle, Lederle Laboratories, Pearl River, N. Y.

A Tissue Vaccine for Hog Cholera—Dr. W. H. Boynton, University of California, Berkeley, Calif.

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*Will be transferred to a general session.

*Some Studies on Bang's Disease—Drs. B. T. Simms and F. M. Bolin, Oregon State Agricultural College, Corvallis, Ore.

The Streptococcic Flora of Dairy Barns—C. S. Bryan, Michigan State College, East Lansing, Mich.

Recent Clinical Methods for Recognizing Nutritional Disturbances—Drs. W. W. Dimock and D. J. Healy, University of Kentucky, Lexington, Ky.

Results in the Use of Fresh and Oxalated Blood of Swine in Making Cellular Counts and Hemoglobin Determinations—Dr. H. C. H. Kernkamp, University of Minnesota, Saint Paul, Minn.

Section on Poultry

Nicotin Sulfate as a Vermifuge for the Removal of Ascarids from Poultry—Dr. Wm. L. Bleecker, University of Arkansas, Fayetteville, Ark.

Some Studies on Infectious Laryngotracheitis of Fowls—Dr. C. A. Brandly, Kansas State College, Manhattan, Kan.

Studies on Fowl-Pox Vaccination—Dr. E. L. Burnett, New York State Veterinary College, Ithaca, N. Y.

Poultry and the Practitioner—Dr. Norman W. Ackerman, Van Horne, Iowa.

Infectious Enterohepatitis of Turkeys—Dr. L. H. Schwarte, Iowa State College, Ames, Iowa.

Some Problems in Poultry Nutrition—Prof. H. L. Wilcke, Iowa State College, Ames, Iowa.

Section on Research and Section on Poultry

JOINT SESSION

Studies on Ulcerative Enteritis in Quail—Dr. J. E. Shillinger, Bureau of Biological Survey, U. S. Department of Agriculture, Washington, D. C.

The Cultivation and Egg-Transmission of the Avian Tubercle Bacillus—Dr. H. J. Stafseth, Michigan State College, East Lansing, Mich.

The Susceptibility of Brucella Abortus and Salmonella Pullorum to Disinfectants—Dr. E. C. McCulloch, University of Wisconsin, Madison, Wis.

Food Requirements of the Larva of Oesophagostomum Columbianum—Dr. R. E. Rebrassier, Ohio Agricultural Experiment Station, Reynoldsburg, Ohio.

*Will be transferred to a general session.

Familial Incidence of Lymphocytoma in Three Generations of the Domestic Fowl—Dr. R. Fenstermacher, University of Minnesota, Saint Paul, Minn.

Preliminary Report of Trichomoniasis of Pigeons—Dr. E. F. Walker, University of Minnesota, Saint Paul, Minn.

Section on Military Medicine

Veterinary Service in Action (Motion pictures)—Maj. F. B. Steinkolk, V. C., U. S. A., Chicago, Ill.

Development of the Veterinary Service in the U. S. Army to 1916—Dr. D. M. Campbell (Lt. Col., Vet-Res), Chicago, Ill.

Experiences of the Late War Should Be Profitable to the Veterinary Corps Now and in the Future—Dr. Reuben Hilty, Toledo, Ohio.

Organization and Function of the Veterinary Service in the National Guard—Dr. S. W. Alford (Capt., V. C., N. N. G.), Lincoln, Neb.

The Inspection Service of the Veterinary Corps—Maj. Edward M. Curley, V. C., U. S. A., Fort Bliss, Texas.

The Clinic

Dogs

Diagnosis of Parasitic and Non-Parasitic Dermatoses—Dr. E. B. Dibbell, Baltimore, Md.

Nutritional Disorders in Pups—Dr. John R. Wells, West Palm Beach, Fla.

Classical Surgical Technic—Dr. Frank C. Mann, Mayo Foundation, Rochester, Minn.

Amputations—Dr. C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.

Dental Anesthesia in Dogs—Dr. E. R. Frank, Kansas State College, Manhattan, Kan.

Tonsillectomy in Dogs—Dr. T. B. Crowe, Chicago, Ill.

Cataract Operation in Dogs—Dr. L. A. Merillat, Chicago, Ill.

Horses

Surgical Treatment of Laminitis—Dr. Glenn L. Ebright, Hammond, Ind.

Surgical Treatment of Hoof Fractures and Contractions—Maj. F. B. Steinkolk, V. C., U. S. A., Chicago, Ill.

Immunization of Horses Against Tetanus—Dr. Ashe Lockhart, Kansas City, Mo.

Administration of Anthelmintics to Horses—Dr. L. N. Morin, McLean, Ill.

Cows

Diagnosis of Mastitis in Cows—Dr. D. H. Udall, New York State Veterinary College, Ithaca, N. Y.

Intravenous Medication in Cows—Dr. T. H. Ferguson, Lake Geneva, Wis.

A New Method of Disinfecting the Uterus—Dr. Otto Stader, Geneva, Ill.

Gastric Lavage in Cows—Dr. Harry Caldwell, Wheaton, Ill.

Epidural Anesthesia—Dr. E. J. Frick, Kansas State College, Manhattan, Kan.

Sheep

Administration of Medicines to Sheep—Dr. F. E. Stiles, Battle Creek, Mich.

A Method of Measuring Fertility in the Ram—Prof. Fred F. McKenzie and Ralph W. Phillips, University of Missouri, Columbia, Mo.

Antiparasitic Medication of the Facial Sinuses in Sheep—Dr. E. E. Slatter, University of Illinois, Urbana, Ill.

Poultry

Collecting Blood Samples for Serological Work in Poultry—Dr. Frank Thorpe, Jr., University of Illinois, Urbana, Ill.

Rapid Blood Tests in Poultry—Dr. E. H. Barger, University of Illinois, Urbana, Ill.

Caponizing—Dr. Frank G. Loomis, Yorkville, Ill.

Diagnosis

Microscopic Diagnosis of Helminthiasis—Dr. Chester D. Lee, Iowa State College, Ames, Iowa.

Blood Examinations—Dr. M. L. Morris, Stelton, N. J.

Urinalysis—Dr. E. C. Khuen, Evanston, Ill.

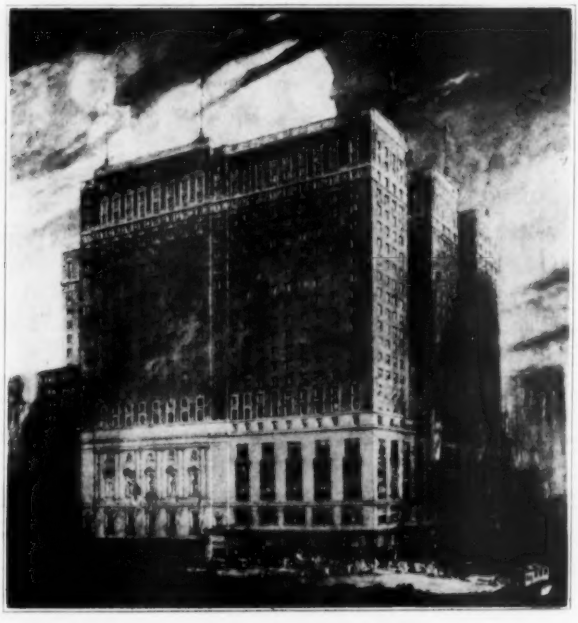
Roentgenography—L. L. Neumann, General Electric Company, Chicago, Ill.

The clinic will be under the general supervision of Dr. L. A. Merillat, Chicago, Ill., assisted by Drs. Harry Caldwell, T. B. Crowe, John B. Jaffray and L. T. Kilfoy, of the Committee on Local Arrangements.

Dr. J. C. Flynn, Kansas City, Mo., will be in charge of the Section on Small Animals; Dr. T. H. Ferguson, Lake Geneva,

Wis., in charge of the Section on Ruminants; Dr. Robert Graham, Urbana, Ill., in charge of the Section on Poultry; and Maj. F. B. Steinkolk, V. C., U. S. A., in charge of the Section on Horses.

Dr. L. Enos Day, U. S. Bureau of Animal Industry, will be in charge of the Section on Microscopic Diagnosis and Dr. R. R. Dykstra, Kansas State College, Manhattan, Kan., will supervise anesthesia.



PALMER HOUSE, CHICAGO

MAKE YOUR HOTEL RESERVATIONS EARLY

Two historic institutions will come together next month when the A. V. M. A. meets at the Palmer House in Chicago—the 70th annual meeting of the national organization and the 62nd year of service for the Chicago hostelry.

The Palmer House, which has been designated official headquarters for the meeting, August 14-18, is outstanding as a convention hotel. The original Palmer House, built in 1871 by Potter Palmer, has given way to a magnificent 25-story struc-

ture, actually a city within itself. But, despite its size, the Palmer House has lost nothing of the fine spirit of hospitality which has characterized it since its beginning. It must be remembered, however, that this is "World's Fair Year" in Chicago, and that the city is host to an unusually large number of visitors from every part of the country. For this reason, it would be advisable for A. V. M. A. members to make their reservations as early as possible.

All of the meetings will be held in the Palmer House, including the clinics—the first time the latter ever have been held under the same roof with the general sessions. The huge Exhibition Hall will house what promises to be one of the most interesting clinics in the history of the A. V. M. A.

For those who are motoring to Chicago, the Palmer House parking and garage service will be of great help. The doormen at the hotel will answer all questions about the service, the charge for which is nominal. There has been established a complete information service for visitors. Courteous clerks are in charge, who will answer all questions about the best way to see the Century of Progress, the city's shops and theaters and other places of interest. The hotel itself is in the heart of downtown Chicago, and the Century of Progress grounds are only a short ride away.

Of especial interest to prospective guests are the rates. These are the same as those established last year. When it is considered that the convenient location of the hotel makes possible a saving in car and taxicab fares, the following schedule is attractive: Single rooms, $3.50 and up; double rooms, $6 and up; suites, $12 and up for parlor and double bedroom. And of its rooms, the Palmer House promises: "All the 2,268 rooms insure the guest quiet, relaxing, comfortable rest."

ETHICAL SERUM PRODUCERS ORGANIZE

With the formation of an organization known as the Associated Serum Producers, Inc., with headquarters at South Omaha, Nebraska, a constructive step has been taken by a score or more of the leading producers of anti-hog cholera serum that should result in considerable benefit to the veterinary profession, particularly throughout the Corn Belt. A publicity campaign has been launched, consisting of a series of carefully prepared display advertisements scheduled for publication in the important

farm papers of Minnesota, South Dakota, Nebraska, Iowa, Missouri, Kansas, Illinois, Indiana and Ohio.

The first advertisement appeared under date of June 10. It was built around the statement, "Only the experienced veterinarian can know both when and how to vaccinate." The second of the series will appear in the same nine publications during July, and will carry the following message: "Vaccination is safe only when backed by science and skill. In unfamiliar hands it may prove a deadly menace." Three other advertisements will appear in these papers at later dates, up to October 1.

A separate series has been prepared and scheduled for the July, August and October issues of *Successful Farming* (Des Moines). This paper has consistently stood behind the veterinarian when others that we might mention have shown a distinctly vacillating policy. During the recent legislative fight in Iowa, *Successful Farming* took a prominent part in defeating the proposal to make Iowa "wide open" as far as the use of virus is concerned. We are glad to be able to make this feeble acknowledgment of the service rendered by Editor Kirk Fox and his associates. They believe that competent veterinary service is a prime essential for a state like Iowa, with such a vast investment in live stock. Furthermore, they believe that it would be a step backward to throw discouragements in the face of the veterinary profession, because Iowa's animal industry would be the loser in the long run.

OHIO ADOPTS LONGER COURSE

Following the lead of four of our veterinary colleges (Iowa, Cornell, Kansas and Colorado), Ohio State University will require a year of pre-veterinary work for matriculation in the College of Veterinary Medicine this fall, according to an announcement recently made by Dean Oscar V. Brumley. The additional year to be included in the veterinary curriculum at Columbus will be made up entirely of general cultural courses, much the same as those required by the other veterinary colleges that have raised their standards. As pointed out by Dean Brumley, the time is here when veterinary medicine must take a step forward in order to be somewhere near equality with the other professions. Five of the ten veterinary colleges in the United States will be operating on the five-year basis this fall.

Come to Chicago—August 14-15-16-17-18

EXECUTIVE BOARD ELECTIONS

The Executive Board elections held in Districts 2 and 3 resulted in returning the present incumbents in both districts by unusually small margins. In District 2, Dr. E. P. Althouse, prominent Keystone State practitioner, beat out the runner-up, Dr. C. J. Marshall, of the University of Pennsylvania, by nine votes. In District 3, only seven votes separated Dr. L. A. Merillat, of Chicago, the winner, from Dr. R. C. Julien, of Indianapolis. Indiana, like Wisconsin, had two candidates in the field and the vote in each of these states was divided. Drs. Althouse and Merillat will succeed themselves at the close of the meeting in Chicago, next month, for five-year terms in each case, expiring in 1938.

CROSSROADS

Within the short space of a few hours, three veterinarians from widely separated points called at the JOURNAL office, on June 19, on various missions. This incident is merely one indication of the fact that "everybody is going to Chicago this summer." The first caller was Dr. G. A. Roberts (K. C. V. C. '03), of Christiansted, Virgin Islands. He was followed closely by Dr. Nelson E. Clemens (Wash. '20), of Hayward, California, on his vacation and in Chicago to see the fair. The third visitor was Dr. Martin D. Baum (Corn. '32), of New York City, en route to the Pacific Coast.

EMPIRE ROOM, PALMER HOUSE, CHICAGO

APPLICATIONS FOR MEMBERSHIP

There has been a very noticeable pick-up in the number of applications for membership received during the past month. As a result, we are able to give first listing to fifteen applications in this issue, the largest number since September, 1932, when the same number were listed. Among the applicants, we are pleased to welcome four 1933 graduates. We hope to see more during the next few months. To join the A. V. M. A. as soon as possible should be the ambition of every one of the approximately 250 veterinarians added to the ranks of the profession at the 1933 commencement exercises in the various colleges. Our correspondence indicates that quite a goodly number are seriously thinking about it and will file their applications just as soon as they get settled.

Do not forget the rules governing the filing of applications. As has been our custom during recent years, we publish every six months an extract from the by-laws of the A. V. M. A. stating how applications for membership should be made. Here is the section giving the information:

> Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Applications must be accompanied by a membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Applications must be filed with the Secretary and examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of the applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BARTLETT, FRED L. 8301 S. Sangamon St., Chicago, Ill.
D. V. M., Indiana Veterinary College, 1921
Vouchers: H. H. Horn and J. V. Lacroix.

BRUNDAGE, HAROLD S. 7417 Stony Island Ave., Chicago, Ill.
D. V. M., Chicago Veterinary College, 1915
Vouchers: E. L. Quitman and L. A. Merillat.

BUTLER, WALTER C. 1813 Belmont, Fort Worth, Texas
D. V. M., Agricultural and Mechanical College of Texas, 1931
Vouchers: Frank R. Jones and Frank D. Porter.

GALLIVAN, FRANK M. Globe Laboratories, Fort Worth, Tex.
D. V. M., Indiana Veterinary College, 1914
Vouchers: Frank R. Jones and H. L. Darby.

GRUTZMAN, MAJOR WALTER R. Box 502, Alachua, Fla.
D. V. S., American Veterinary College, 1896
Vouchers: J. V. Knapp and James G. Fish.

KLINE, ALBERT J. Wauseon, Ohio
V. S., Ontario Veterinary College, 1894
Vouchers: J. H. Lenfestey and E. M. DeTray.

MCCLEAD, FORREST R. 4, Alexandria, Ind.
D. V. M., Ohio State University, 1933
Vouchers: W. F. Guard and Jas. D. Grossman.

MCKITRICK, JOHN L. Station B, R. 1, Columbus, Ohio
D. V. M., Ohio State University, 1933
Vouchers: W. F. Guard and Jas. D. Grossman.

MARKLE, WILLIAM M. West Newton, Pa.
V. M. D., University of Pennsylvania, 1933
Vouchers: Louis A. Klein and G. A. Dick.

MILLER, SHEFFORD S. 300 Webster Ave., New Rochelle, N. Y.
D. V. M., Ohio State University, 1932
Vouchers: W. Reid Blair and Frank H. Miller.

PINNEY, FREEMAN 6421 Harper Ave., Chicago, Ill.
M. D. C., Chicago Veterinary College, 1910
Vouchers: R. S. MacKellar and G. E. Totten.

RICKS, DANIEL H. Pauls Valley, Okla.
D. V. M., Alabama Polytechnic Institute, 1930
Vouchers: C. C. Hisel and L. J. Allen.

SMITH, KENNETH F. 2129 Peasley St., Columbus, Ohio
D. V. M., Ohio State University, 1933
Vouchers: W. F. Guard and Jas. D. Grossman.

TRUSSELL, WILLIAM E. Charles Town, W. Va.
B. V. Sc., Ontario Veterinary College, 1932
Vouchers: C. T. Higginbotham and H. M. Newton.

WEBB, HAROLD A. 7417 Stony Island Ave., Chicago, Ill.
B. V. Sc., Ontario Veterinary College, 1928
Vouchers: E. L. Quitman and L. A. Merillat.

Applications Pending

SECOND LISTING

(See June, 1933, JOURNAL)

Ahrens, George A., 6351 Normal Ave., Chicago, Ill.
Bradham, Guy E., Cisne, Ill.
Corliss, James R., State House, Augusta, Me.
Held, Vincent, 1504 Oregon Ave., Klamath Falls, Ore.
Meginnis, Paul J., 6421 Harper Ave., Chicago, Ill.

The amount which should accompany an application filed this month is $7.50, which covers membership fee and dues to January 1, 1934, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. July 5, 1933. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 6-7, 1933. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Interstate Veterinary Medical Association. Elks Building, Omaha, Neb. July 10, 1933. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.

Washington State Veterinary Medical Association. Olympia, Wash. July 10-12, 1933. Dr. Clifford Ackley, Secretary, Winlock, Wash.

Ontario Veterinary Association. Ontario Veterinary College, Guelph, Ont. July 11, 1933. Dr. H. M. LeGard, Secretary, 335 N. Main St., Weston, Ont.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 12-13, 1933. Dr. E. A. Caslick, Secretary, Paris, Ky.

Maine Veterinary Medical Association. State House, Augusta, Me. July 12, 1933. Dr. L. E. Maddocks, Secretary, R.F.D. 2, Augusta, Me.

Tulsa County Veterinary Association. Tulsa, Okla. July 13, 1933. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Mississippi Valley Veterinary Medical Association. Peoria, Ill. July 13, 1933. Dr. Lyle Gray, Secretary, Bushnell, Ill.

Vermont Veterinary Medical Association. Brandon Vt. July 13-14, 1933. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

Virginia State Veterinary Medical Association. Fredericksburg, Va. July 13-14, 1933. Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

Western New York Veterinary Medical Association. Alleghany State Park, N. Y. July 13, 1933. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Northeastern Indiana Veterinary Medical Association. Tri-Lakes, Ind. July 16, 1933. Dr. H. W. Demsey, Secretary, Huntington, Ind.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. July 18, 1933. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Missouri Veterinary Medical Association. Joint meeting with Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. July 18-19, 1933. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

South Carolina Association of Veterinarians. Aiken, S. C. July 18-19, 1933. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. July 19, 1933. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

New Jersey Veterinary Medical Association. Hotel Madison, Atlantic City, N. J. July 20-21, 1933. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Northwestern Ohio Veterinary Medical Association. Hotel Harding, Marion, Ohio. July 20, 1933. Dr. W. P. S. Hall, Secretary, Division of Health, 9 Ontario St., Toledo, Ohio.

Montana Veterinary Medical Association. Bozeman, Mont. July 27-28, 1933. Dr. Hadleigh Marsh, Secretary, Agricultural Experiment Station, Bozeman, Mont.

Saskatchewan, Veterinary Association of. Regina, Sask. July 27-28, 1933. Dr. Norman Wright, Secretary, 137 20th St. West, Saskatoon, Sask.

Connecticut Veterinary Medical Association. Bridgeport, Conn. August 2, 1933. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

Poultry Science Association. Michigan State College, East Lansing, Mich. August 2-5, 1933. Professor C. G. Card, General Chairman, East Lansing, Mich.

Hudson Valley Veterinary Medical Society. Rhinebeck, N. Y. August 9, 1933. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

American Veterinary Medical Association. Palmer House, Chicago, Ill. August 14-18, 1933. Dr. H. Preston Hoskins, Secretary, 1230 W. Washington Blvd., Chicago, Ill.

Illinois State Veterinary Medical Association. Palmer House, Chicago, Ill. August 15, 1933 (8:00 p. m.). Dr. J. V. Lacroix, Acting Secretary, 1817 Church St., Evanston, Ill.

Wisconsin Veterinary Medical Association. Palmer House, Chicago, Ill. August 15, 1933 (1:00 p. m.). Dr. B. A. Beach, Secretary, Madison, Wis.

Fifth World Poultry Congress. Trajan's Markets, Rome, Italy. September 6-15, 1933. Comitato del V Congresso Mondiale di Pollicoltura, Ministero dell' Agricoltura delle Foreste, Roma, Italy.

A STUDY OF BRUCELLA INFECTION IN SWINE AND EMPLOYES OF PACKING-HOUSES*

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The epizootiological data with respect to Brucella infection in swine, based chiefly on positive agglutination tests, that have accumulated during the past three years are a fair criterion of the distribution of the disease in swine in the United States. The accuracy of the agglutination test as a means of measuring the presence of the disease in a herd cannot be questioned. When a bacteriological examination is carried out on reacting hogs, *Brucella suis* can be isolated from one or more tissues of a considerable number.

Systematic epizootiological surveys of the disease in a large number of hogs in many herds already have been made by Hardy and associates,[1] Murray and McNutt,[2] and Johnson and Huddleson.[3] The results of these surveys are compiled in table I. These surveys reveal considerable variation in the percentage of infection in individual herds. In the survey which we made, the degree of infection, based on an agglutination titre at 1:50 or above, varied from zero to 23 per cent of the total number in each herd.

The bacteriological data thus far accumulated indicate that *Br. suis* is the only species of the genus capable of infecting the hog naturally. It has been conclusively demonstrated that this species of Brucella is infectious for man, and that the infecting strain is giving rise to a symptom-complex not measurably unlike that due to *Brucella melitensis*.

From our present knowledge of the disease in the hog on farms, the important question that naturally arises is: How important is the infected hog from a public health standpoint?

It appears to us that an answer to this important question might be obtained by conducting the necessary examinations of hogs slaughtered for meat consumption in packing-houses and

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of men who work in different sections of the houses. The examinations should be conducted by those procedures which will reveal the percentage of Brucella-infected hogs slaughtered for meat and whether meat preservation processes employed in the average house have any effect upon the viability of *Br. suis* in infected hog meat. Further, the necessary examinations should be conducted on the employes of the packing-houses to detect evidence of present and possible past infection.

There already has been accumulated considerable evidence as to the status of Brucella infection in both hogs and employes in packing-houses in widely separated sections of this country. In table II are compiled the serological findings of Hardy and associates,[4] Boak and Carpenter,[5] Murray and McNutt,[2] and Gwatkin,[6] on hogs slaughtered at packing-houses. The percentages of infection found in the slaughtered hogs, by the four groups of investigators, vary considerably. But regardless of this variation, there is presented ample evidence of the fact that infected hogs are arriving at slaughter-houses and are being passed for utilization as meat or meat products.

TABLE I—*Comparison of results of the agglutination test conducted on hogs on farms in several states.*

REPORTED BY	STATE	HERDS	HOGS TESTED	RESULTS OF AGGLUTINATION TEST			
				< + 1:40		> + 1:40	
				No.	%	No.	%
Hardy *et al*	Iowa	60	611	96	16	109	18
Murray and McNutt	Iowa	64	560			132	23.5
Johnson and Huddleson	Michigan	17	2,251	390	17.3	313	13.5

Hardy, Hudson and Jordan,[4] in searching for epidemiological evidence of undulant fever in a packing-house in Iowa, examined the blood of 217 employes for specific agglutinins and found 37 (17 per cent) reacting below a titre of 1:80 and 30 (14 per cent) reacting in a titre of 1:80 or above. The percentage of workers reacting to the agglutination test in a dilution of 1:80 or above was directly proportional to the opportunity to contract infection through the handling of fresh hog tissues.

Martin and Meyers[7] examined the blood of 200 packing-house employes for evidence of infection, 100 of which had been on the company's sick list at some period within the 18 months before

the agglutination test was made. They found 15 per cent of those on the sick list and 3 per cent of the other employes showing Brucella agglutinins above a 1:40 dilution. Of the 15 cases with a history of previous illness, three had been diagnosed as undulant fever. The remaining twelve presented symptoms suggestive of undulant fever. It is interesting to note that the three individuals with no history of illness showed agglutination titres above 1:100. This finding is important, because it is further evidence of the fact that many individuals, who come in contact with infective material, never show clinical symptoms of the disease. This is particularly true of veterinarians, as was shown by two of us,[8] and by Haxthausen and Thomsen,[9] in Denmark.

TABLE II—*Comparison of results of the agglutination test conducted on hogs slaughtered at several packing-houses.*

REPORTED BY	STATE	HOGS TESTED	RESULTS OF AGGLUTINATION TEST			
			< + 1:50		> + 1:40	
			No.	%	No.	%
Hardy *et al*	Iowa	343	98	28	74	21
Boak and Carpenter	New York	4,014			64	1.5
Murray and McNutt	Iowa	1,278			34	2.6
Gwatkin	Ontario	500	4	0.8	3	0.6

The purposes in mind in conducting the studies reported in this paper were to add to the data presented in table II and, in addition, to determine bacteriologically the extent of the presence of the organism in the tissues of the reacting hogs; to study the effects of meat preservation processes used in packing-houses on the viability of the organism present in hog tissues; and to determine the extent to which employes are exposed to infection through the handling of infected hog tissues.

Part I—Serological and Bacteriological Findings in Slaughtered Hogs

METHODS AND RESULTS OF STUDY

The hogs examined in this study were slaughtered in one large and one small house in Michigan. The large house slaughtered chiefly hogs shipped from the Chicago market, while the small

one obtained many of its hogs from farms in Michigan as well as the Chicago market.

The blood of the hogs was examined directly after slaughter for the presence of Brucella agglutinins, by means of the rapid agglutination test. The test was conducted by mixing one drop of antigen with a large drop (about 0.04 cc) of whole blood on a glass slide with a tooth-pick. The mixture was tilted back and forth for one-half minute and the reaction read by holding the slide directly in front of a bright light. From previous experience we have found that the whole-blood test, when conducted in the manner just described, will detect agglutinins in a 1:50 dilu-

TABLE III—*Summary of the serological and bacteriological findings on hogs examined at packing-plants in Michigan.*

EXAMINATION	HOGS TESTED	HOGS REACTING	REACTORS (%)	BR. SUIS ISOLATED	BR. SUIS FROM REACTING HOGS (%)
1	286	11	3.84	4	36.36
2	158	4	2.53	1	25.00
3	149	8	5.37	1	12.50
4	93	8	8.70	0	0.00
5	257	22	8.56	0	0.00
6	193	12	6.21	2	16.66
7	191	12	6.28	0	0.00
8	138	5	3.62	1	20.00
9	288	15	5.20	2	13.33
10	241	14	5.80	2	14.28
11	247	5	2.02	0	0.00
12	268	11	4.10	0	0.00
13	183	16	8.74	3	18.75
14	118	3	2.54	2	66.66
15	272	12	4.77	4	33.33
16	220	15	6.81	5	33.33
17	227	35	15.41	3	8.57
18	204	56	27.45	4	7.14
19	201	8	3.98	0	0.00
20	41	36	87.80	7	19.44
Totals	3,975	308	7.74	41	13.31

tion or above. All of the recorded positive tests indicate a reaction in a 1:50 dilution or above.

Those hogs showing an agglutination reaction with whole blood were marked in such a way that they could be identified easily

when they arrived in the eviscerating line. From the reacting ones were removed the spleen, a large piece of liver, the kidneys and the following lymph-nodes: gastric, internal iliac, supramammary, mesenteric and bronchial. The tissues were brought to the laboratory and cultured within six hours after removal from the hog. The culturing was carried out by smearing the cut surface of the tissues over the surface of liver-agar plates containing gentian violet in a 1:200,000 dilution. The inoculated plates were incubated aerobically and in CO_2 for seven days before they were discarded as negative.

All suspicious colonies of *Br. suis* appearing on the plates were transferred to liver-agar slants and identified later by the agglutination test. Each culture was classified as to species by dye differential media and H_2S production.[10]

The results of these studies are summarized briefly in table III. The two packing-houses were visited on 20 separate occasions at wide intervals between June, 1931, and January, 1932. The results obtained on the total number of hogs tested on each occasion are tabulated separately. The percentages of reactors found on the separate examinations vary from 2.02 to 87.80. Only three groups (17, 18 and 20) show a relatively high percentage of reactors. Groups 17 and 18 were Iowa hogs purchased from the Chicago stock-yards. The hogs in group 20 were from a Michigan farm. We were aware of the presence of infection in this herd from previous blood tests.

When the hogs were slaughtered, they were grouped into light and heavy grades. The native (Michigan) hogs were killed in a separate group. This point was kept in mind at the time of each test. The blood test results, according to the origin and the type of hog, are recorded in table IV. It is quite evident that the heavy or older hog is more likely to react positively than is the light or younger hog. This point will be discussed later.

The percentage of positive isolations from reacting hogs corresponds very closely to the results of similar examinations made on naturally infected hogs killed on farms in the state of Michigan. The probable reason for such a low percentage of isolations in reacting hogs has been mentioned in a previous paper.[11] The location of *Br. suis* in the tissues of the 41 hogs in which it was found is summarized in table V. Here again results are presented which parallel previous cultural findings from the bacteriological examination of naturally infected hogs.

Part II—The Effect of Meat Preservation Processes on the Viability of Br. Suis in Naturally Infected Hog Tissues

Methods and Results of Study

The average packing-house makes a practice of chilling the hog carcass at a temperature of 32 to 34°F. for 48 hours after it is dressed. The length of time of holding the meat and internal organs varies considerably and depends largely on the retail demand. As a rule, hog liver is not retained in storage for more than four or five days before it is sold to the retail butcher or made up into liver sausage, which is later smoked and cooked in hot water. There are times when fresh hams, bacon and shoulders are kept at a temperature of 18 to 20°F. for as long as six months before curing and smoking.

Table IV—*Summarized comparison of results of test on native (Michigan) and Chicago rough and light hogs.*

Source	Type	Hogs Tested	Reactors	
			No.	%
Native	Light	1,052	52	4.91
Chicago	Light	2,616	194	7.41
Native	Rough	197	52	26.3
Chicago	Rough	110	10	9.0

In studying the effect of cold on the viability of *Br. suis* in naturally infected hog tissues, it was thought desirable first to maintain the tissues at an extremely low temperature. If it was found that the organism did not survive at this temperature, then the effect of a higher temperature would be studied. The first temperature at which the infected tissues was studied was −10°F. The results obtained on three different types of tissue at this temperature are recorded in table IV. Certain pieces of tissue, such as lymph-nodes, were so small that it was impossible to make more than two bacteriological examinations after different intervals of exposure.

In most instances there was a slight decrease in the number of colonies growing out on the plates as the interval of exposure to cold increased. The decrease appears to be directly proportional to the number of colonies found on primary examination. Although the total number of colonies obtained on each examina-

tion by this method is a very rough representation of the actual number of organisms present in the tissues, the number of different examinations made is sufficient to show conclusively that there is no appreciable decrease in the number of organisms for the first ten days of incubation at −10°F. Further, when the organism is present in the tissues in large numbers, a considerable percentage of them survive for 40 days. Several attempts were made to determine the effects of higher temperatures, such as 0°F. and 10°F., but the tissues which were used were discarded after the incubation in the cold was begun, due to the fact that the primary cultures failed to show the presence of the organism.

The other meat preservation method employed in this study was that of "salt-curing brine." It is a common practice in many packing-houses to cure hams, bacon and shoulders in a solution of certain salts for 25 to 30, 12, and 12 to 15 days, respectively, at a temperature of 38°F. When the curing process has been completed, the meat is "smoked" for 18 hours at a temperature of 110°F.

The tissue which was selected for this study was the spleen of the hog. It is realized that this tissue is not utilized by the packer for edible purposes, but the results obtained from a study of the effect of the curing process on the organism in the spleen should furnish evidence of what will happen to the organism, if present, in glandular tissue in a ham treated in the same manner.

The curing brine used in this study was made up for us at one of the packing-houses from the formula employed by the house.

The spleens from many reacting hogs were cultured and immersed in the brine and placed at a temperature of 40°F. Those spleens from which no primary isolations were obtained were discarded later. Small sections of those spleens that were positive culturally were removed from the solution at the end of five, ten, 20, 30, 40 and 45 days of incubation and cultured according to the previously described technic.

The results obtained from the study of eight infected hog spleens are summarized in table VII. The tabulated cultural findings indicate that the mixture of salts in solution, used for curing ham, bacon and shoulders in these trials, may render *Br. suis* in infected spleens non-viable or considerably reduce it in numbers at the end of a five-day exposure. The chance of recovering *Br. suis* from the spleen, as indicated by the number of colonies appearing on the plate after exposure, appears to depend

on the number present. If there are only a few organisms found present before exposure in the brine, very few, if any, will be found alive at the end of five days of exposure. On the other hand, if they are numerous in the beginning, the tissue still may contain viable organisms after 45 days.

TABLE V—*Location of Br. suis in tissues of 41 naturally infected slaughtered hogs.*

TISSUE	HOGS GIVING POSITIVE CULTURE
Spleen	18
Gastric lymph-gland	8
Internal iliac lymph-gland	7
Supramammary lymph-gland	6
Liver	5
Kidney	4
Mesenteric lymph-gland	3
Bronchial lymph-gland	2

Part III—The Examination of Employes of Packing-houses for Evidence of Brucella Infection and Exposure to Brucella

METHODS AND RESULTS OF STUDY

Two methods or tests were employed in seeking evidence of infection or exposure to Brucella in the employes of packing-houses. One was the classical serum agglutination test and the other involved the measuring of the opsono-cytophagic activity of whole citrated blood for Brucella.

The blood specimens were collected in 10-cc amounts in Kidel tubes containing sufficient sodium citrate to prevent clotting and to obtain a final dilution of 0.8 per cent of the salt in the blood.

The rapid method was employed in examining the serum for Brucella agglutinins. The maximum dilution used was 1:500.

The opsono-cytophagic test for detecting those exposed to Brucella will be described and discussed in detail in a forthcoming paper.

A total of 167 employes in four packing-houses and one stock-yard were examined. Some effort was made to obtain evidence of a past history of clinical undulant fever, but owing to the conditions under which the blood samples were collected, only a few employes were questioned in this matter.

The development of a clinical case of undulant fever in one of

the packing-houses, about eight months after the first test was made, necessitated the retesting of 18 of its employes.

The results of the blood examinations are summarized in table VIII. Of the total number examined, 18 (10.7 per cent) showed agglutinins for Brucella in their blood serum and 40 (23.9 per cent) gave evidence of having been exposed to the organism in some manner according to the phagocytic activity test. In house 1, two men, one a veterinary inspector, were, at the time of the test, showing periodical clinical symptoms characteristic of undulant fever. The agglutination titre in one case was 1:100 and in the other 1:500. The phagocytic activity test indicated active

TABLE VI—*Summary of results from keeping infected hog tissue at −10° F.*

TISSUE	COLONIES OF BR. SUIS				
	ON PRIMARY ISOLATION	CULTURE FINDINGS AT END OF			
		5 DAYS	10 DAYS	30 DAYS	40 DAYS
Supramammary lymph-gland	60	58	45		
Gastric lymph-gland	15	11	12		
Mesenteric lymph-gland	3	Neg.	Neg.		
Liver 1	2	Neg.	Neg.		
Liver 2	5	3	1	Neg.	
Spleen 1	81	32	65	61	53
Spleen 2	12	7	3	3	1
Spleen 3	Numerous	Num.	Num.	Num.	84
Spleen 4	11	10	7	6	4
Spleen 5	7	Num.	Num.	Num.	Num.

infection in both cases. One employe in house 2 gave a clinical history of previous infection. No information was obtained as to past or present infection in the other employes examined except in a retest of a group in house 1. This group of men was employed in the sausage manufacturing department. All in the group were negative to both tests when made in November, 1931. We were asked to make another test on this group in July, 1932, due to the fact that one of the group became severely ill from undulant fever. The clinical symptoms in this case were typical of the disease and the blood serum showed an agglutination titre of 1:1000. Shortly after this employe became ill, three others in the group showed mild symptoms of the disease. Of the total number examined, including the one who had been ill for five weeks, the serums of 11 (61 per cent) agglutinated Brucella in

dilutions above 1:25. The phagocytic activity test was made on the blood of 16 men of the group. The polymorphonuclear cells of five of these showed activity of the degree seen in active infection and seven showed activity of the degree seen in immunity resulting from exposure to Brucella. It was not possible to measure the phagocytic activity of two individuals, whose serums showed an agglutination titre of 1:500, owing to the clotting of the blood after collection. Three members of the group whose cells showed a very marked phagocytic activity, which is characteristic of immunes, were negative to the agglutination test. *Br. suis* was isolated from the blood of one of the mild cases. The two tests together furnish evidence that 14 (77.7 per cent) of the 18 examined had been exposed to Brucella in recent months to a degree great enough to have caused either immunity or infection.

TABLE VII—*Summary of results from keeping infected hog spleens in curing brine.*

Tissue	Colonies of Br. Suis						
	On Primary Isolation	Culture Findings at End of					
		5 Days	10 Days	20 Days	30 Days	40 Days	45 Days
Spleen 1	6	2	2				
Spleen 2	2	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Spleen 3	30	8	9	5	5	2	1
Spleen 4	4	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Spleen 5	13	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Spleen 6	21	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Spleen 7	3	2	5	Neg.	Neg.	Neg.	Neg.
Spleen 8	52		43	6	6	2	

DISCUSSION

The serological and bacteriological data presented in the first part of this paper show quite conclusively that hogs infected with *Br. suis* are being slaughtered for meat in Michigan packing-houses. The percentage of infected animals killed daily, based on the serological findings, may vary from 2.3 to 87.8 per cent. The number of reacting hogs in which the presence of the organism could be demonstrated culturally varied from zero to 66.6 per cent. The total number that were positive culturally represents 1.03 per cent of the total number of hogs tested.

In view of the fact that the degree of Brucella infection varies

TABLE VIII—*Summary of blood examinations on packing-house employes.*

Packing-House	Employes Tested	Number Showing Agglutination Reaction						%	Exposed or Infected (Indicated by Phagocytic Activity)		
		Negative	1:25	1:50	1:100	1:200	1:500		Infected	Exposed	Total (%)
1	29	19	2	3	4	0	1	34.5	2	8	34.5
2	10	6	1	1	1	0	1	40.0	0	4	40.0
3	18	15	2	0	1	0	0	16.6	0	3	16.6
4	72	71	1	0	0	0	0	1.3	0	15	20.8
5	38	38	0	0	0	0	0	0.0	0	10	27.3
Totals	167	149	6	4	6	0	2	10.7	2	38	23.9
1*	18	7	1	2	1	0	7	61.0	5	7	66.6

*Retest.

from farm to farm, one would expect a considerable variation in the percentage of reacting hogs that are slaughtered daily at the houses. Then, too, if it so happens that only light hogs are killed on the day or days the test is conducted, one may expect to find the percentage of positive reactors much smaller than what it would be if only heavy hogs were slaughtered.

The manner in which the bacteriological examinations were conducted possibly furnishes only approximate information as to the presence or absence of the organism in the reacting hog. It is realized that when one cultures a small section of the spleen from a reacting hog and finds the organism absent, the negative findings do not necessarily mean that it is not present in another section of the spleen. On the other hand, the reverse may be true if the results obtained in Part II of this paper, from the repeated culturing of infected spleens kept in a frozen state, have any significance. The data indicate that if the organism is present in one section of the spleen, it is present in other sections as well.

The number of colonies of *Br. suis* that appear on the surface of plates inoculated with hog tissues varies considerably. Often only two or three colonies may be found, but occasionally the surface of the plates is completely covered with a heavy growth of the organism. The latter type of growth was obtained from sections of two livers and several spleens.

The infected organs were examined very closely for gross changes which might be useful in detecting the disease such as one sees in the organs of infected guinea pigs, monkeys and goats. There were often seen lymph-nodes considerably congested and raised hemorrhagic areas distributed over the gastric surface of the spleen. These changes were observed as often in non-infected hogs as in those that were infected. It is very doubtful whether any particular significance can be attached to the above-mentioned gross changes in so far as the Brucella-infected hog is concerned.

It is interesting to note that the locations of *Br. suis* in infected hogs slaughtered at the packing-houses correspond very closely to our previous findings in infected hogs slaughtered on Michigan farms. It is of further interest to note that *Br. suis* was the only species of the genus recovered from the infected hogs.

The data that were obtained from holding naturally infected hog tissues at $-10°$F. indicate that the viability of *Br. suis* is little if any affected over a period of 30 days. There is some

evidence that it is reduced in numbers when the tissues are held at the temperature indicated for a longer period.

In the experiment to determine the effect of curing brine solution on the viability of *Br. suis* in naturally infected hog spleens, the cultural results obtained would indicate that the present practice employed in many packing-houses, of holding or curing hams, shoulders and bacon in brine for 12 to 30 days, is an effective method of destroying the organism should it be present in the meat in small numbers. On the other hand, if there is a large number of organisms in the tissues, a few may be found alive after exposure for 40 days in the brine solution.

Since a considerable amount of hog meat is used in the manufacture of different types of pork sausage, we thought that it would be of considerable interest to know whether *Br. suis* could be found alive in sausage after its manufacture had been completed.

The sausages chosen for this experiment were of five types, namely: whole pork, frankfurter, ring Bologna, raw liver and cooked liver. All of the sausages, except the first, after being smoked for three hours, are cooked under water at a temperature of 160 to 180°F. for 20 minutes to one hour. The length of time they are cooked depends upon the diameter of the sausage.

The cooked liver sausage is made of cooked liver, pork and beef tripe. It is pasteurized again in sausage casings made from the ceca of sheep. The raw-liver sausage contains hog liver and pork. It is cooked in the casings in hot water after being smoked at a temperature of 180°F. for 45 minutes and then at 160°F. for 30 minutes.

The sausage meat mixtures just mentioned were heavily inoculated with a saline solution suspension of *Br. suis* by means of a hand spray and then pressed into casings. They were then smoked and cooked according to the procedure used in the packing-house. Inoculated uncooked specimens were kept for controls.

The sausages were cultured in the same manner as described for culturing hog tissues. Unfortunately, the control specimens were so contaminated with *B. coli* that it was impossible to detect the presence of *Br. suis* in them by cultural methods. The cooked sausages appeared to be sterile culturally, that is, in so far as Gram-negative organisms were concerned.

Even though all of the cultural results on the sausages were negative, they may be said to have considerable significance. Since the cooking process completely destroyed *B. coli* present in the uncooked sausage in great numbers, it would be difficult to

conceive of *Br. suis*, if present naturally in the sausage, surviving the same treatment.

The results from the blood examinations of packing-house employes confirm the results of a similar study conducted by Hardy and associates,[4] Jordan,[12] and Martin and Meyers,[7] as regards exposure to, or active infection with, Brucella. We are using the term "exposure" in the sense of the passage of the organism through the epithelial barrier of the skin or mucous membrane.

The results of the agglutination test in our study do not furnish a true picture of the number of employes that have been exposed to infection. On the other hand, we feel certain that the results of the phagocytic activity test are more indicative of the extent to which packing-house employes are exposed to Brucella infection as a result of handling infected meat. The extent of exposure depends largely upon the nature of the work of the employes. In one house the blood of only one of 25 women engaged in wrapping cured bacons showed a degree of phagocytic activity seen in exposed individuals. In the same house the blood of each of the ten men working in the "offal" section showed a marked phagocytic activity. The agglutination test was negative in all cases.

Since the examination[8] of veterinarians in this State for possible Brucella infection by means of the agglutination and intradermal tests revealed that a considerable percentage of those who engage in cattle practice have been exposed to infection without showing clinical manifestations characteristic of the disease, it has been our contention that when exposure to Brucella takes place (passage of the organism through the epithelial barrier), the individual develops either an active immunity or active infection which manifests itself clinically. If either of these states can develop in exposed veterinarians and also in laboratory workers engaged in studying the disease, they must also occur in individuals who constantly handle infected meat. The opsono-cytophagic test used in this study beyond a doubt furnishes additional evidence of the truth of our contention.

The importance of infected hog meat as a means of conveying undulant fever to the consumer cannot be answered from the results of the foregoing studies. About the only possible manner in which the consumer could become infected is through the handling of infected meat before it is cooked. There is as yet no epidemiological evidence to indicate the seriousness of this possibility.

SUMMARY

A total of 3,975 hogs slaughtered at two packing-houses in Michigan have been studied for the presence of Brucella infection. Of the total number examined by means of the whole-blood rapid agglutination test, 388 (7.74 per cent) showed agglutinins in their blood. From 41 (13.31 per cent) of the reacting hogs, *Brucella suis* (Traum) was recovered from one or more tissues.

The maintenance of hog tissues naturally infected with *Br. suis* at a temperature of −10°F. for 30 days has little if any effect upon the viability of the organism.

When hog spleens naturally infected with *Br. suis* are kept in meat-curing brine solution for as short a period as five days, it is very difficult to recover the organism by cultural methods. The organism may be recovered in small numbers in heavily infected spleens after being in brine for a period of 40 days.

The data from the examination of 167 employes in packing-houses indicate that 10.7 per cent, according to the agglutination test, or 23.9 per cent, according to the opsono-cytophagic activity test, have been exposed to Brucella from the handling of infected hog tissues.

The results from the retesting of 18 employes in one packing-house, after the occurrence of a definite clinical case of undulant fever would indicate that when exposure to infective material takes place, the exposed develop active immunity or active infection.

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STERILITY IN CATTLE*

By W. A. BARNETTE, *Greenwood, S. C.*

What is sterility? Sterility is the inability to reproduce young. What are some of the apparent common causes of sterility in the cow? The apparent common cause of sterility in the cow primarily, in most cases, is infection, which leads to one or more of the following: vaginitis, cervicitis, endometritis, pyometra, salpingitis, hydrosalpinx, pyosalpinx, oöphoritis and cystic ovaries. Other causes in some cases may be emaciation, ovarian tumors, tuberculosis or senile atrophy of the generative organs. Diseases of the generative organs causing sterility concern us most in cattle kept for reproduction and dairy purposes.

In field and everyday practice, we may classify factors leading to sterility in cattle under three general heads: (a) feeding; (b) care before and after calving; (c) family breeding, whether in-breeding or line-breeding.

In feeding cattle it is not uncommon for the owner and interested parties to work with only one end in mind—production. Certain results are expected from the ration set up. The procedure is carried on and no bad effects are realized until breeding problems appear. Then the entire schedule must be changed in order to care for reproduction.

To mention briefly what happens quite often in feeding, it may be said that the constant feeding of high percentage protein feed, improperly balanced with vitamins, minerals and the best of hay, results in production that may be satisfactory for a period, but finally reproduction becomes a problem, as the system has been so disturbed and changed that the reproducing organs do not function normally. No attempt is being made to outline or mention all of the pitfalls in feeding which may lead to sterility in cattle. We are aware of the fact that a working knowledge of feeding is necessary. When sterility is to be treated, aside from the corrections which the veterinarian may make by various operations or manipulations, if we hope to get results, he must be able to help all concerned by checking the ration and, if necessary, outline the feed program for the herd and especially the individuals under treatment.

Without a question, the most trying times on cattle are the periods of a few months before and after calving. Mechanical or organic invasions must be guarded against in every way

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possible. Premature calving and improper care may bring on any of the common causes of sterility. One of the most frequent causes is neglect and improper care of retained afterbirth and a general check on the reproductive organs. Continuous neglect of service a reasonable time after calving, when normal heat periods occur, is another. To enlarge on the common cause of sterility, which is primarily due to infection after calving, would take too long. It is understood that those who discuss this paper will handle a few of these cases in detail, giving outlines of care, handling and treatment.

Family breeding of cattle has led to some of the family weaknesses being handed down, resulting in sterility. Sterility of this nature is hard to correct. For instance, when a breeder is following in-breeding, each time he breeds, the chances are, if his selections are not made with care, the offspring will inherit at least one-half of the weakness on either side and if this weakness should be in reproduction it will be more difficult to overcome, the further the breeding is carried on. The same would be true in line-breeding, but with one-fourth the danger.

Discussion

Dr. W. K. Magill: We find that we have a great deal of trouble not only in the mature cattle but in heifers, and the question as to when to treat or when not to treat is quite often brought up. I usually ask an owner of an animal to breed her at least three, or possibly four. times to a bull which we know is potent before we attempt to treat her.

In treating, I usually go in and make a thorough examination of the vagina, the cervix, the uterus, and the ovaries. We try to determine whether cervicitis, metritis, or any other changes have taken place in the ovaries. Quite often we go into animals, particularly heifers—we find sterility more commonly in heifers than in mature animals—when they are coming into heat normally, and on examination we can determine no change. We might class that more or less as a functional disturbance. I remove the corpus luteum. Some say it should not be done, but I get results, and so I do it.

I have in mind one heifer that I treated. Many times heifers will abort at about the third or fourth month. Quite often, after they are bred, they will immediately come back in heat. I advise the owner to hold them over for about sixty or ninety days, and they usually will breed from then on. This heifer that I have in mind had a little age on her, and she had been tested for abortion. When she aborted she was again tested and she failed to react. From that time she has bred annually. She would conceive, carry a calf about four months, and then abort it. She was held over until her fourth heat period—they usually come in heat about the second or third day after they abort—then this animal bred and dropped a nice calf. Today she has the state record in Class G in Guernsey cattle.

In the mature cow, of course, we have a great deal more to contend with than we do in the heifer, because we know that at one time she was a breeder. When we go into such a case, we can expect to find more trouble, and it is up to the practitioner to determine the cause. Quite often that is difficult. I will admit myself that very often I go

to see an animal and there is practically no cause at all that I can determine, although I know that there always is a cause. We know that there is a cause for *this* animal not breeding. It is necessary, then, to go into the history, whether she calved normally, and whether she had any trouble following her calving. Then make your examination to determine whether the cause is from chronic metritis, cervicitis, retained yellow bodies, cystic ovaries, or what not. Quite often we find a slight discharge. I remove the yellow bodies. That is the chief thing that I do when I cannot find a cervicitis or chronic metritis. Of course, in chronic metritis, we treat with douches and in chronic cervicitis I use Lugol's solution, swabbing the cervix about every fifteen to eighteen days until it returns to normal, although I will say that from my results in treating chronic cervicitis, particularly where there is laceration, it takes a long, long time to bring them around.

In massaging ovaries or removing yellow bodies, care always should be taken not to injure the fimbriated portion of the tube, because if you do, you are very apt to have some adhesions that probably will lead to closure of the tube following the treatment.

In most of these cases, where on examination you find apparently no cause—where the uterus, the cervix, and the vaginal canal are normal—you may find a little discharge, and you may not, but invariably you will find a very prominent yellow body or an enlarged ovary. Sometimes you cannot break them down and have to go back again. That has to be done at period suitable to you and to the owner. I usually go in again in about a week to break down the ovary because it must be broken down until you leave nothing but the normal ovary tissue there. If you have a large one, particularly with age on it, you are going to have some trouble bringing it down to normal. The biggest trouble that I have is getting the farmers to start early enough, because so often they will carry an animal along for a year and mention the fact to you that she is not a breeder. Then I will say that I will be glad to treat her at any period, or when she is past the period. You cannot go in there immediately after she has been served because you cannot determine pregnancy under eight to ten weeks. I never go in until I know that she is past the heat period or that she has been in heat. I had a case of a cow which, the owner kept telling me, would not conceive, would not breed. When she was eight years old, she dropped a calf without ever being treated. In a pure-bred business an animal like that is not profitable to carry, but I am bringing that point out to show you that it is not advisable to go into an animal at any time unless you know she has passed a heat period. Once in a while you do find one that comes into heat even though she is pregnant, but that is very, very uncommon.

Dr. C. H. Case: With my herds I have tried to use preventive medicine. To prevent sterility rather than treat it, I have induced my herd-owners to let me make monthly visits to their herds and to check over every cow. They pay me monthly and expect me on a certain day. I have 32 herds that I visit every month, some large and some small. The largest has 325 cows. I keep a loose leaf record book. These cows are numbered, and the names are put down if they are pure-breds. I put down the breeding date and the calving date and all records of examinations and work done on each cow. When I go into the barn, I check over every cow. If there is a cow that has not been bred, I examine her to see what is wrong, to see whether she has a cystic ovary or a corpus luteum retained. I always inquire whether cows have been discharging pus. If they have, I douche them.

I carry my books with me in my car. I write down every detail before I leave the place, so that if anything comes up and a client

asks, "Did you take care of that cow?" I can look in my book and know exactly what has been done.

Since I have been keeping these records, they have proved to be valuable more than once. For instance, one of my men said that when I removed the corpus luteum from a sterile cow there was no use breeding her the first time. I always make the remark that they will come in estrum three to ten days after the corpus luteum is removed. He said, "No use. Very few of them conceive." I took my books on five herds and went over my records of 214 cows. Sixty-six, about one-third, conceived, on an average, in about 15 days; 67, another third, conceived in an average of 25 days, and the other one-third on the next heat period, an average of 46 days. Where a corpus luteum is removed, about one-third will conceive on the first breeding. I examine all the cows at the end of two months to see if they have conceived.

The dairy that has an ideal record, as Dr. Williams tells us, is the one in which every cow has a calf every twelve months. I have one herd that averaged one calf every eleven and one-half months. That is the first and only herd to make such a record. If you keep track of the records, you will find that it is quite a job to have a perfect dairy.

Bang's disease makes lots of trouble, including sterility, but we also have sterility in our clean herds just the same, quite a little of it. We are going to have sterility in all herds on account of heavy feeding to make large milk-production. Of course we all know that a cow which gives birth to a calf in eight months or seven months, and which has a retained placenta, is going to take two or three months before getting back into shape to breed. She is going to lose three or four months of production more than the healthy cow.

I have a client who wants a cow to have a calf in August or February, because he is going to show at fairs. A calf born before September 1 is classed as a yearling at fairs, although it may be almost two years old. We have dairymen who hold off for the records. Then we have forced feeding to get these large records, and they hold off breeding. I have one Guernsey cow now that is in the ninth month. She is going to make close to 1,063 pounds of butter fat. That is going to be a real large record in our section, and she is not bred yet. They are holding her off because as soon as she becomes pregnant she will drop on her production of milk. The Holstein people do the same thing. I think the double letter class, which the Guernsey people have, is one of the greatest things we have to cause sterility. There are very few cows that have enough constitution to breed regularly after making a record. For instance, we had one cow in one of my herds that gave 149.2 pounds of milk in 24 hours when milked four times a day. She holds the world's record. Since she made that record, however, she aborted a calf at five months. She is negative to the blood test, and we are having trouble in getting her to conceive again. It just destroyed the equilibrium of breeding. We have another heifer, however, a two-year-old, that gave 120 pounds of milk and averaged 94 pounds of milk for three months. She was never under 60 pounds of milk in nine months, although that heifer bred again and is due to calve in a short time. Those are the things that make trouble and cause sterility.

Infected bulls may cause cows to abort and become sterile. In handling my best pure-breds I demand that they wash out the sheath of the bull before and after every service. I do this because in some herds we have had lots of trouble, and I know that the trouble came from the bull.

As to the ordinary sterile cow which you are called upon to treat, you can get the best results when she is in estrum. We examine the

ovaries first for a cyst or retained corpus luteum, and make sure that we get that corpus luteum out. If you cannot get it by expression through the rectum, you generally can remove it with one hand in the vagina. Be sure that you get it out, because you have not done that animal any good if you do not. Then I douche the uterus of every cow. Any man who does sterility work and does not wash out the uterus of every cow showing signs of sterility is guilty of collecting money while he is not giving the owner good service, because so many of those cows have pus in them which cannot be found by external examination. I have come to the conclusion that the fewer times you put your hand into the vagina of a cow, the better off the cow is. I use a speculum and a forceps and never put my hand into the vagina to douche the uterus. You can carry infection from one farm to another. In years past, when I used to use the ordinary Williams forceps, I would put my hand into the vagina to catch hold of the neck of the uterus. The cows never showed any discharge, but two or three days later, they would show a discharge of pus from the vagina, due to infection being carried into the vagina by the hand and arm. I had to do something else. I either had to quit that which I know had to be done, or else I had to find some new way. The speculum and forceps were the result.

The first six months of this year we douched the uteri of 345 cows. We averaged over 100 cows every month which we examined for pregnancy, to be sure they were safe with calf. That is the way we work to try to prevent sterility.

The worst thing we have to deal with is cystic ovaries. I wish somebody would find out something about what causes cows to develop cystic ovaries. I have found that in about 90 per cent of the cows that have cystic ovaries I find pus in the uterus. A great many cows with cystic ovaries, when broken down, return with a cystic ovary month after month. If you keep breaking them down and washing them out until you get the uterus clean, quite a number of them will breed, but the owner gets discouraged. Unless it is a pure-bred animal, he cannot afford to keep her, becauses she will not produce milk and he has a loss. In douching these cows I use sterilac, 10 grams to 1 gallon of water. You can use anything you like. If you use a chlorin solution, I think you will get better results. If, on the return flow, these cows show any pus, I inject 10 cc of mecurophen. I make a 1:1,000 solution and use a 10 cc syringe with a long nozzle. I inject it down in the uterus, through a catheter, and leave it there. I have had a great deal better results since I have used mecurophen than I ever had before.

DR. J. F. DEVINE: It seems as though we probably have made some progress in this work, but I just cannot see how we can give much to men here in a few minutes, because to the men who are actually doing the work, the men who have become interested during the past 15 or 20 years, anything that we might say here would be very academic, and inexperienced men would not get very much good out of it, if we are not academic.

I have talked, as you know, at many of your meetings for the last 15 to 20 years, and each time I try to stress the fact that if a man wishes really to learn something about breeding troubles—I do not know why we ever called it sterility; if an animal is sterile it is sterile and done for, at least that is what we think about other things—of course, he should make a special effort to learn the normal anatomy of these organs, even more thoroughly than he learned at school, and there is no way that I know, of becoming proficient at the work except by constant practice.

Then again, the young man of today is confused. One so-called authority will tell him one thing and another, another thing, and

it is a good deal like Pat said to Mike, "One man's word is just as good as another's," and Mike said, "Yes, and a damned sight better."

I remember the time when we used grappling tongs on cervices, grabbing them, and infecting them, and some of our so-called experts told us that we should irrigate every heifer. I recall following up these men in herds of pure-bred cattle, where irrigating had been practiced at every estrual period. Such work usually gave someone else a job for several months after.

Then another man will tell you that you should never use instruments. Of course, when people become such extremists, so erratic, you do not get anywhere. All this holds true with the mares.

I see Doctor Baird here. Doctor, in 1913, I went down to your country and spent a couple of weeks there. I worked on several farms with three or four veterinarians. I now recall one particular breeder. He had 23 mares which had been giving him trouble for two, three or more years. We examined those mares and I condemned one of them outright—an old senile thing, 22 years old. I classified two as questionable. I felt they might be made to breed if they could have the proper attention. In those days I had no laboratory facilities. This is the point that I want to stress: Those two cases of endometritis—they scarcely could be styled pyometra—had chronically infected flaccid uteri. If I had had a laboratory and had taken cultures, as we would today, I probably would have condemned both of them. I, of course, do not know positively. I would depend on what was found. But I put them down in the doubtful list. The story in the end was that one of those, with no treatment but massaging, bred promptly, and the other never did breed. Today, if we were to examine those mares, we might condemn both of them with our present knowledge of bacteriology, so we must be careful and not make erratic decisions.

Following, or at about that time, Doctor Dimock began the work and he has perfected the bacteriological technic and is doing fine work. I think he is the best man on equine breeding troubles in America, if not the world. As to medicines. You cannot disinfect a mucous membrane. Go to any of your nasal or oral surgeons and they will tell you that it cannot be done. You can disinfect it if you literally burn it, but there is no other way. You can stimulate the normal germicidal secretions the same as they do when they irrigate the sinuses with a normal saline solution. If it were not for the normal secretions, we would have no healthy females, because they are eternally exposed to infection.

That brings up another thing, particularly in cattle. Almost every herd now and then has a retained placenta. That means that there is a catarrhal exudate passing around that stable no matter how sanitary it may be. The cow gets it on her tail and slaps it along to her neighbor, with the result that if you go into a herd with the best of sanitation, say 100 head of cattle with 15 or 20 in trouble, probably, 70 per cent of them need nothing but vaginal douches for a week or two before breeding; such cases are readily cleaned up. So, the young man will say, "What are you going to do for this or that?" The first thing is that he must know the normal from the abnormal, and the thing that I have eternally stressed is to be careful and do no harm. As a matter of fact, from the different methods that are being recommended, it looks as though it does not matter what we do if we do not do any harm. Gentle manipulating, and careful and regular douching seem to have merit. Sometimes, if one has had a good bit of experience, he may be able to do something for cases that may appear hopeless.

For instance, some ten or twelve years ago, a man in Kentucky bought a cow for $8,000 at a sale in Buffalo, in June. She was due

to calve in July. Up to the following March she had not calved; I finally went down to see her. We took from that cow about 22 quarts of pus. Both horns were filled with pus simulating twin pregnancy, with the exception, of course, that pus is lighter than a fetus and the uterus was not so low as it would be if weighed down with a fetus or two fetuses. Notwithstanding the fact that this cow had been in this condition for nearly two years, the only thing we had to do was to empty her uterus. Then everything floated right up into the pelvic cavity. After her uterus was emptied she was irrigated, and it was massaged out. In three or four days, I came back and massaged it again. About a week later, before I left, I massaged her again. There was not an adhesion and no salpingitis. If there had been a salpingitis, or adhesions, then the results might have been different. She had two normal heat periods at regular intervals. On the second period she was bred, and conceived. She went on and bred for years.

Conversely, there is the danger of an inexperienced man going into herds and aborting cattle. That is a serious offense. A stock-owner calls him in as an expert, because the animal perhaps has been bred several times and has not conceived, and without being familiar with the symptoms of pregnancy, he may reach the ovary, perhaps if she is not too far advanced, take out the corpus luteum, and in three or four days a calf is dropped. It is too bad. If you want to learn this thing you must make an examination at every opportunity you can. You will not hurt a cow if you examine her, if she is suffering from other things, unless she is extremely sick. So, in addition to taking advantage of every opportunity to examine the reproductive organs in the living animal, it is well to do the same thing at abattoirs until you have seen the variety of conditions that may occur. A very helpful way is to examine the animals before slaughter and again after.

DR. JOHN BAIRD: Gentlemen, if I may, I would like to speak of the condition we have in mares. When I came to this country, I knew absolutely nothing of sterility troubles in the mare. We, in Britain, have sadly neglected this subject in the mares, and it is only within the last few years that members of the British profession are getting alive to the importance of the subject.

It did not take me long to realize that I must improve my knowledge on the subject of sterility, and as I had eight or ten mares which had been barren for several years when I took charge of them in 1928, I naturally wanted to know why they were not breeding. I consulted Dr. Dimock, and the knowledge I have derived from his tuition has been most valuable, and I am now able to diagnose an infected mare, from clinical examination, but generally take a culture at the same time, to make sure of my clinical diagnosis.

These mares all had streptococcic infection, and the subsequent treatment was most unsatisfactory. Every modern antiseptic irrigation of the uterus was tried, but with absolutely no results. I treated them for the best part of two years, and as they did not clean up, they were disposed of.

I may be wrong, but I am of the opinion that treatment is of no avail. Time and nature will clean up the majority of cases. If a mare is a producer of high-class stock, the owner can afford to leave her alone for two years, and not breed her until she has cleaned up. If she is only a moderate producer, get rid of her at once, and herein lies the value of being able to make a correct diagnosis—economy in stud fees and keep of moderate mares.

As regards ovarian trouble. All I am able to do, is to diagnose a normal or an abnormal ovary. In 1931, I had six mares which kept

in season all the time. These mares were all free from streptococcic infection, so I asked Dr. Dimock to examine them for me and, with your permission, I will give you a short history of each mare:

Mare 1: Eight years old; first foal in 1930. Barren, 1931. In season practically all the time in March and April of 1931. Examination showed both ovaries to be cystic. These were punctured. Mare bred in 1931 and 1932 but did not get in foal, so she was disposed of.

Mare 2: Six years old; first foal, 1930; barren, 1931. Both ovaries cystic and punctured. Was bred about a month later, conceived and foaled in 1932 and again in foal for 1933.

Mare 3: Three years old; maiden. Right ovary very much enlarged; punctured. Bred a few weeks later; conceived and foaled in 1932 and again in foal for 1933.

Mare 4: Ten years old; foaled last in 1930. Punctured both ovaries. She did not get in foal and was disposed of.

Mare 5: Fifteen years old; barren for two years. Punctured both ovaries. Conceived and foaled in 1932, but now barren.

Mare 6: Twelve years old. Had four foals in succession, the last in 1930. In the breeding season of 1930, she came in season regularly every 21 days from March until the end of May. I did not, at this time, worry about this mare not getting in foal. In the spring of 1931, she took the horse at the commencement of the breeding season, and never went out at any time. Examination revealed both ovaries to be cystic. They were punctured and the mare not bred in 1931. In 1932, she acted as in 1931 and would take the horse at any time, but did not get in foal. I will give this mare another chance in 1933, but am positive that her breeding days are over.

I talked with Dr. DeVine the other night about ovarian trouble, and I gather from his remarks that he is more in favor of massage treatment.

Before I sit down, may I tell you of the great compliment paid to Dr. Dimock, in 1931, when he was asked to go over to England to investigate sterility in several large Thoroughbred breeding establishments. His work and findings were so satisfactory, that he is again over in England at the present time, and it looks as if he will have an annual visit, in connection with sterility work. This, to you members of the American Veterinary Medical Association must indeed be pleasing.

New York Broadcasting Program

Under the auspices of the Committee on Broadcasting, New York State Veterinary Medical Society, the following talks are scheduled over Station WGY (General Electric Station) during July and August:

July 14—"Poultry Pointers (When the Hen Gets Sick)," Dr. J. R. Starkey, Goshen.

July 28—"Maintaining Public Confidence in Milk," Dr. L. J. Tompkins, Sheffields Farms Co., Inc., New York.

August 11—"The Veterinarian and the Health Officer," Mr. E. R. Gauhn, Monroe County Department of Sanitation, Rochester.

August 25—"Johne's Disease Paratuberculosis," Dr. John J. Wermuth, Warwick.

The time of these broadcasts is 12:30 p. m., E. S. T.

LAMENESS AND ITS TREATMENT IN THE HORSE*

By JOHN BAIRD, *Coldstream Stud, Lexington, Ky.*

In presenting the subject of lameness in the horse, it is not my intention to infer for one moment that I can put before you anything original, or that I am an authority, but from experience I have realized that it is difficult at times to feel conscientiously satisfied that one has given a correct opinion in a case of lameness. I have always been keenly interested in treatment, and although I have not been in active practice for several years, and have not kept notes, I will endeavor to relate some of my experiences.

GENERAL TREATMENT OF LAMENESS

The exciting causes of lameness vary greatly in nature and severity, and although each case should be treated according to its particular requirements, there are certain curative measures which can be applied more or less satisfactorily in all cases.

Wounds and bruises causing lameness should be treated by surgical methods. After a wound or bruise has healed, lameness may continue, owing to the effects of the injury interfering with function, as the movement of a joint, the play of a tendon or ligament, or with muscular action.

Acute or chronic inflammation, of bone, ligaments, tendon or muscle, is the predominant cause of lameness, and in most cases treatment must be directed to arresting or modifying the inflammatory process. The age of the patient, position and nature of the lesion or cause, should be considered before treatment is commenced.

In all cases attention should be given to the feet and shoes. Removal of one or all four shoes may be desirable, but in some cases the shoe must be removed to facilitate examination and treatment of the lame foot. Lameness caused by corn, seedy toe, or sand-crack usually requires immediate alteration of the shoe.

Lameness due to acute sprain of the perforans tendon or subcarpal ligament, spavin, or curb, is benefited by a thick-heeled shoe, as the raising of a heel affords relief by supporting the hind part of the foot and easing the tension of the flexor tendons.

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*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

In perforatus strain a high-heeled shoe is not advisable, as it tends to induce knuckling at the fetlock.

Rest is one of the most valuable aids to the successful treatment of lame horses. For many it is imperative, for the majority it is beneficial, and for all it is desirable. Warm fomentations are beneficial in painful lameness, but the employment of hot water in a satisfactory manner is frequently attended with inconvenience. To succeed, warm fomentations should be repeated frequently at short intervals and suitable bandages applied to prevent evaporation and too rapid loss of heat. Hot fomentations, by dilating the vessels at the periphery of the injured part, relax the tissues, ease pain, and promote rest. They are indicated especially in cases of wrenched joint, severe contusion, and muscular sprain. When infection of the part is suspected, a non-irritating antiseptic should be added to the water.

Lotions and liniments are often employed in the early stages of lameness. Caution is required in applying liniments to inflamed parts, for often they are used at the wrong time and aggravate the mischief they are intended to repress. Cold in the form of douching, spraying or continuous irrigation with cold water is often valuable in the initial stage of inflamed tendon, joint or muscle. Recent sprains are much benefited and readily prepared for more active measures, by refrigerant lotions of alcohol and water, solution of ammonium chlorid, lead acetate, of Epsom salt.

Massage or hand-rubbing is very useful in limb cases, and generally in a downward direction, but a better result would follow rubbing or kneading upwards, or in the direction of the venous blood-stream. Usually it is desirable to prescribe a bland oil to be well rubbed into the swollen joint or tendon, and many recent sprains of tendons have been completely removed and the lame horses made sound, by this means.

Counter-irritation, by blistering or firing, often is employed, but too much should not be expected from either. Nothing can be gained by firing a sidebone or an established ring-bone; the ossified cartilage is beyond repair, and firing has no influence for good on the joint already fixed by anchylosis.

Neurectomy is often quite useful in the treatment of chronic lameness of the lower part of the fore or hind limb, after counter-irritation has failed. Chronic lameness arising from navicular disease, ring-bone, or exostosis of the fetlock can be removed by resection of nerves, and in suitable cases this operation should be performed without hesitation or delay.

Shoulder Lameness

Shoulder lameness is necessarily a comprehensive term, because many of its examples are indefinite as to nature and causation. Cases in which there is no evident alteration of the part very rarely admit of positive diagnosis. In most cases, movement of the scapula is restricted, it appears held to the trunk, the step is shortened, the joint is raised insufficiently, and the foot—apt to catch the ground—is swung outwards, or dragged, or it may be carried.

Certain forms of shoulder lameness present additional symptoms which may be characteristic and of considerable assistance in diagnosis. In suprascapular paralysis, and owing to loss of power in certain muscles which support the joint, the point of the shoulder appears to roll outwards when the foot comes to the ground, and within a few days of the inception of the lameness, the extensor muscles become plainly atrophied, producing noticeable deformity of the shoulder. In arthritis, the point in locomotion is fixed, the limb is carried, and sooner or later the point of the shoulder becomes enlarged.

Treatment: In simple cases of shoulder lameness, rest and a mild blister will generally effect a cure, but I have not found this treatment to be sufficient in cases of atrophy. In such cases, I have found that the intramuscular injection of turpentine is most successful. It seems to act as an intramuscular blister. Five cubic centimeters of turpentine can be injected at multiple points over the scapular region. In a few minutes there is intense irritation. In 12 to 24 hours, a large swelling surrounds the area involved, and most of the irritation has gone. This swelling may disappear in a few days, or it may persist and fluctuate. If fluctuation is marked, the swelling should be opened, when quantities of thick, creamy fluid, resembling pus in appearance, will exude. In a week or ten days, the lesion will have practically resolved, and a marked improvement will soon be noticeable.

Arthritis

Arthritis of the knee, sometimes referred to as "knee spavin," in causation, symptoms and treatment, much resembles the corresponding condition occurring in the hock joint. When the disease is confined to the lower part of the joint, movement may be only slightly interfered with. Lameness in the early stages is always accompanied by marked swelling of the knee and the formation of a considerable amount of fibrous thickening. Later,

this in great part disappears, and a hard and bony swelling appears.

Treatment: Hot fomentations, two or three times daily; dry thoroughly, massage, and rub in a lotion of a refrigerant nature, such as one consisting of ammonium chlorid, lead acetate and alum, one ounce each, dissolved in a pint of water.

Soak bandages in this lotion and apply them to the affected knee. Secure them in position by a linen or preferably a rubber bandage. Later on, it may be necessary to blister, and if this does not give relief, median neurectomy may be resorted to.

Sore Shins

Sore shins is a form of periostitis affecting the metacarpal bones of young horses, particularly race horses, and is due to the fact that they are called upon to perform an amount of work at an age when the condition of their bones is totally unfit to bear it. Sore shins may affect the whole shaft of the bone, and may be so excessive as to cause necrosis to supervene, but usually the inflammation is circumscribed and confined to the lower extremity and anterior portion of the bone. If the subperiosteal exudation is very great, and the swelling involves a large extent of the surface of the bone, the most active treatment may be employed in order to prevent necrosis.

Treatment: Cold applications are indicated in the early stages, either in the form of cold-water bandages, or the hose pipe. If the hose-pipe is used, apply it twice daily, for 30 minutes at a time, then dry the leg thoroughly, and soak a large piece of cotton in acetate of lead, sulfate of zinc and alcohol. Apply over the affected bone and then cover with a bandage. When inflammation has subsided, it is advisable to blister, if for no other reason than to prolong the rest treatment.

Splints

Splints are another form of ostitis and periostitis affecting the metacarpal bones, and in rare instances the metatarsals. They have a great analogy to sore shins, differing only in situation; splints being usually found upon the inner surface of the leg, involving the inner small and large metacarpal bones, whereas the other form usually is found only on the lower third of the large bone. The reason for this may be on account of the fact that the pace of the race horse is the gallop, in which the weight of the body is thrown upon the anterior portion of the bone with fearful velocity at each successive bound, causing the inflamma-

tion of the bone in that part in which the shock is received. Splints, on the other hand, are caused by a moderate pace, where the animal is more kept up to the bridle, and the weight thus thrown more directly upon the upper extremity of the bone.

Treatment: The majority of splint lamenesses can be cured with any good absorbent, and very few cases require the firing-iron.

A good splint dressing may be made as follows: Dissolve four drams of bichlorid of mercury in three ounces of alcohol. Add two drams of tincture of iodin and two ounces of turpentine. Rub in daily, until a scurf appears. Complete rest, of course, is essential.

SESAMOIDITIS

The posterior part of the sesamoid bones is covered with cartilage, forming a groove, over which the flexor perforans plays. Both the cartilaginous pad and the tendon are liable to injury, which causes severe and obstinate lameness. This is always a serious lameness, and treatment is very unsatisfactory. A horse, after a period of rest, may trot quite sound for a short time, but is liable to fall lame again with moderate work.

Fracture of the sesamoids is frequent in race horses, and of course it is useless to expect complete recovery, to enable a horse to undergo strenuous work.

Treatment: You are all familiar with ring-bones, sidebones, pyramidal disease of the os pedis, and navicular disease, and treatment for these is very unsatisfactory. In these diseases, where there is marked contraction of the foot, good results often are obtained from sawing up the foot. I believe you Americans call it "grooving." In Britain, we use a fine-toothed, double-edge cutting-saw, but I have been unable to get this instrument from any of the makers here.

Begin at the center of the foot, right under the coronary band, make the incision with the saw, straight down to the solar surface, and keep sawing until you come to the sensitive laminae. Then make three or four similar incisions on each side of the foot. Keep the horse standing in poultices for several days in order to assist in softening the foot. Allow the patient to go barefooted for at least three weeks. This allows expansion of the foot. Then put a light bar-shoe on and insert springs. An ordinary horse may be put to farm work in three or four weeks, but with race horses I insist on rest for four months before beginning training, and at the end of this time you will be surprised at the increase in the size of the foot. Many cases re-

cover to carry on work and race successfully for many years, after undergoing this treatment. As a last resort, neurectomy, the lower operation, may be resorted to. I have always refused to do the higher operation.

May I relate one particular case which came under my care last fall? A two-year-old Thoroughbred filly, with naturally small, boxy feet, contracted at the heels, had been in training for three months. She was sent home by the trainer to be fired for osselets on the ankles. She was not actually lame, but there was a lack of freedom in her action. The feet had been neglected and the heels were much too high. I came to the conclusion that, with correct shoeing, the filly would get sound and remain sound. (Incidentally, I will say that I consider a very, very large percentage of lamenesses are due to faulty shoeing.) However, I decided to operate on this filly, with the object of getting expansion of the foot and a wider heel.

Before beginning to operate, I had the heels lowered and the toes shortened. I did not have a saw, so improvised the flat firing-iron, and made the grooves as I have already explained. For about a week afterwards, the filly was very sore, and stood in poultices all the time. At the end of the third week, I put a light bar-shoe on, inserted springs and blistered the coronary bands. Then I turned her out in the paddocks every day during the winter, and saw that the heels were rasped down when necessary. At the end of a month, when all soreness from the operation had subsided, the filly was absolutely sound, and in June of this year, when she went into training, the heels were exactly one inch wider than they were before the operation in October. I saw the filly recently, and she has never shown any symptoms of soreness.

LAMINITIS

Treatment: Adrenalin is advocated in the early stages, injecting it over the course of the plantar nerves. In theory this is correct treatment, for if one can diminish the volume of blood flowing into the foot, it is natural to expect that the total volume of blood will be diminished. But I have not had great results from this treatment in acute cases, and so far have not found anything to excel a very old-fashioned treatment, *viz.*, bleeding from the point of the frog. Before doing this, I thin the sole as much as possible, shorten the toes and, with a sharp knife, make a free incision at the point of the frog. Blood will flow freely from the incision, and I immediately put the foot in a warm bran poultice, to which a reliable antiseptic has been

added. At the same time I give a dose of physic. In about six hours after bleeding, I put the foot into a hot antiseptic bath for about half an hour, and when the foot is taken out of the bath, I renew the poultice. This treatment is repeated several times daily. In about 24 hours, or after the physic has acted, there is a marked improvement, and it is rare to have dropped sole with this treatment.

Canker of the Foot

It has been recorded that the dominant infection in this trouble seems to be the bacillus of necrosis. To permit the infection to become active, there must be some small wound, or wounds, in the horn. These wounds may be various, and I have seen cases starting in the center of the sole as a result of a bruise. You are all, no doubt, familiar with the symptoms of canker, so I shall now briefly relate a treatment with which I have been most successful.

Treatment: Put the horse under a general anesthetic, remove all diseased horn, and be most particular that all diseased horn is removed. At this stage, it is wise to use a good tourniquet. Reduce the foot to normal size, pare down the healthy horn to the thinnest. Then, if the edge of the hard horn is lifted with the forceps, one can remove it, as far as it is underrun, with the knife. It is better to remove a small edging of sound horn all around the area. The bar is most likely affected and this must be completely removed, and similarly with the frog, except where sound. Be careful, if the diseased process extends out to the wall, to determine the limits of its work. If you have much pathological growth, remove it with scissors and, after it appears cleaned up, apply a dressing which must thoroughly disinfect the part. At the same time, the foot must be kept as dry as possible.

My favorite dressing is nitrate of lead, combined with formalin, and I will say that I have never come across a case which did not yield to this treatment.

The dressing is made as follows: Put four ounces of flour and two ounces of nitrate of lead into a mortar and mix them thoroughly. Add two drams of formalin and enough mineral oil to make a soft putty paste. Smear this dressing liberally over the affected area, cover with cotton and bandage, and put a strong covering of leather over the sole. Then put on a shoe and a plate. Renew the dressing every third day, taking care to scrape out the old dressing before applying fresh paste.

INJURIES TO THE FLEXOR TENDONS AND SUSPENSORY LIGAMENT

Treatment: The most important point is to stop work. Fresh cases, due to mechanical injury, are treated the first 24 to 48 hours by cold applications, but after this, little good results from the use of cold. I then prefer moist warmth, supplemented by proper bandaging, applied as follows: Two long rolls of cotton, somewhat thicker than a man's thumb, are moistened and laid lengthwise on both sides of the diseased tendon, close to the metacarpus, and fixed there by a linen or cotton bandage, wetted and applied rather tightly. Over this, a dry woolen bandage is placed, so as to exercise constant pressure on the material below and on the diseased spot. When available, a piece of rubber tissue may be placed between the two bandages. This dressing is changed every four hours. The moist warmth and regular pressure favor reparative changes; excessive extravasation and cellular proliferation are checked, and resorption assisted.

Careful massage also is beneficial and, with the above-mentioned treatment, will cure and remove the swelling in cases where, with the antiphlogistic methods formerly employed, thickening almost always remained.

The slight vascularity of tendons in no way supports the theory of the action of cold; on the other hand, moist warmth and methodical compression assist absorption, and are certainly not so likely to impede repair as cold. My experience shows this treatment to be by far the best.

The action of blisters is due largely to the mechanical effect of infriction and the pressure of the cutaneous thickening on the inflamed spot; and when the above-described treatment fails, it is good practice to apply a blister, in the form of a blistering plaster. Immediately after applying the blister, cover the leg from the knee to the fetlock with a good thick layer of cotton, and over this apply a bandage. Leave the bandage on for two or three weeks, and see that the skin at the back of the knee and heels does not crack.

BOG SPAVIN

Treatment: Disinfect the hock with a 10 per cent carbolic lotion before injection. This acts also as a slight local anesthetic. Use an ordinary 10-cc syringe with a fine needle about 1½ inches long. If the hock is very tense, insert the needle into the center of the swelling, and let out what synovia will run out. Do not draw off any more fluid than will run out of its own accord

through the needle, assisted by a little pressure of the fingers on the swelling. Inject anything from 10 to 30 cc, depending on the size of the hock.

The animal will show considerable lameness and swelling of the hock for 7 to 21 days. I find that horses vary a lot. In most cases, the swelling will have disappeared in from three to four months, some much sooner. If operating on a working horse, I find that the best results are obtained by working the animal as soon as the lameness subsides. The injection is useful also in stifle lameness with enlarged joints, and also in capped hocks and wind galls.

The prescription I use consists of one part of antipyrin, one part of spirits of tannin and ten parts of alcohol.

Diseases of the Joints

Synovitis: By the term synovitis is indicated an inflammation of the synovial membrane. It may be either simple or acute, or it may be purulent or suppurative. In the simple form, there is little or no tendency for the affection to implicate the other structures of the joint, whereas in the suppurative form the joint capsule, the ligaments and the bones soon come to participate in the diseased process, giving us a condition known as acute arthritis.

Acute simple synovitis is nearly always brought about by injury to the joint, or by sprains of the ligaments.

Treatment: If a joint has been injured by blows or other causes—in other words, if the injury is subcutaneous, and no wound is in existence—there is no treatment which offers better results than does the continued application of cold. When the case is a marked one and the animal valuable, benefit will be derived from the application of crushed ice. The condition of the animal must be watched, and the case helped as far as possible by the administration of a mild dose of physic, by saline drinks and, when necessary, by small and repeated doses of tincture of aconite in order to relieve the pain. In a chronic case, the repeated application of a blister is indicated.

In suppurative synovitis, we have synovitis complicated by the presence of pus. Unlike the simple form, it shows a marked disposition to spread, and quickly involves the surrounding structures.

Arthritis: With an attack of simple synovitis, it always may be assumed that the changes commenced in the synovial membrane have communicated themselves more or less readily to

the surrounding tissues, and are not confined to the synovial membrane alone. We thus may have the inflammatory phenomena asserting themselves in the surrounding ligaments, in the bone, and in the articular cartilages. It depends, in fact, upon the severity of our case whether we call it synovitis or arthritis. The two conditions merge so frequently that no hard and fast rule may be laid down whereby they may be differentiated with certainty.

Treatment: You are all familiar with the symptoms, diagnosis and pathology of these diseases, so there is no necessity for me to take up time in describing them. It is treatment with which we are most concerned. Inasmuch as many of these cases have their starting point in penetrating wounds, we shall be concerned first with the correct treatment to be adopted when we know the wound to have reached the articulation. Poultices should never be applied. Warm antiseptic baths are much to be preferred, as the solution readily comes in contact with the deeper parts of the wound, but it is questionable, however, whether even the bath is sufficient. We have in this case a deep punctured wound that in every probability is infected with the organisms of pus. The most rational treatment, therefore, in the early stage is to irrigate the wound freely with a solution of carbolic acid in water (1:20) injected by means of a glass syringe. This injecting should be done thoroughly, and the opening plugged with a piece of cotton, soaked in a solution of iodoform and alcohol, and an antiseptic pad placed over all. The joint should be bandaged and, in the case of the foot, encased in a protective boot. The bandage should be removed daily and the wound redressed.

Once the suppurative process is checked, it is good treatment to blister the whole region of the joint smartly with cantharides and biniodid of mercury. This prevents further infection of the wound to some extent.

Before concluding, I should like to say a few words about firing. When I became familiar with conditions in the racing stables in America, I was very much surprised to see the large number of high- and low-class horses bearing marks of the firing-iron, many for no obvious reason. In my inquiring why, prominent trainers told me that they considered firing strengthened the legs, and some even went as far as to say that it prevented or materially assisted a horse from breaking down. In their arguments in favor of firing, they were supported by many prominent veterinarians, who instilled into their minds the fact

that promiscuous firing was beneficial. These veterinarians have built up a lucrative business from the practice of firing and, from a commercial point of view, one cannot blame them. But looking at it from a purely professional aspect, I ask you if it is correct treatment. Most emphatically, I say it is not.

Take, for instance, a bunch of yearlings which have been broken in July and kept in light training for three months or so. They come out of this training in a sound condition. The suggestion comes from the owner or trainer that these sound colts should be fired in order to toughen and strengthen the legs. The veterinarian has no alternative but to do as he is asked, for if he does not, some other one will. But my point is this: Has the veterinary profession not reached the stage when the members could try to educate horsemen against the fallacy of this absurd treatment? The idea that normal ligaments and tendons can be strengthened by the use of the firing-iron is too ridiculous to be considered.

As for firing spavins, ring-bones, or sidebones, I have yet to see the case where one could conscientiously say that firing effected a permanent cure, and I make bold to say that better results will be obtained from correct therapeutic treatment.

From the foregoing, you are not to conclude that I would never advise the use of the firing-iron. In the case of an old, indurated tendon, I might be tempted to use it, but if this tendon had been correctly treated at the time of injury, there would have been no occasion for the iron.

This, gentlemen, concludes my remarks. Many of you may not agree with me, and I hope there will be a good discussion, especially on firing.

Horse Prices Hold Their Own

The farm price of horses during the period from December, 1919, to December, 1932, declined only 27 per cent, while prices of all farm products declined 61 per cent, according to the U. S. Department of Agriculture. The price of mules declined 34 per cent during the period. In December, 1932, the farm price of horses averaged $56.

Farm population increased over a million during 1932. The figures, as of January 1, 1933, were 32,242,000, compared with 31,241,000 on January 1, 1932, according to the U. S. Department of agriculture.

FURTHER OBSERVATIONS ON BULLA-OSTEOTOMY IN THE DOG, WITH CASE REPORTS*

By J. E. McCoy *and* G. W. McNutt

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The following further observations have been made on bulla-osteotomy in the dog since we first reported.[1]

Two instruments have been added to the list mentioned in our first paper as necessary in bulla-osteotomy: Ralk's finger-nail drill and Luer's curved bone-rongeur. When the stage of operation is reached in which the bulla ossea is exposed, the periosteum may be scraped from the bone conveniently with the finger-nail drill. Then a small opening is made through the center of the bulla to form a "seat" for the plain-tooth drill. Then the latter is used to enlarge the opening, after which any shreads of mucous membrane may be removed with a small forceps. The Luer bone-rongeur is now extremely convenient to enlarge the opening to any extent desired.

While we have been using the Mayo retractor, another on the same order but one which may be held open and secured at any desired width, is in the process of manufacture. With the standard pattern, it sometimes is necessary to stretch the tissues too much in order to reach a notch at which the Mayo instrument may be secured.

Copper or other metal ear-applicators are valuable in cleaning and treating the external ear. Other types of applicators are too large for this work.

We have designed and are having made specula which conform to the measurements of the external acoustic meatus of dogs, and find that they are a wonderful improvement over the old ones and aid materially when making examinations for diagnosis. We also are having alligator forceps made that are long enough and small enough to be worked through these specula.

Epinephrin is of value in checking any hemorrhage which might be produced while cleaning and examining the external acoustic meatus.

Of the five cases reported below, three made perfect recoveries. We have every reason to believe these will be permanent. Two cases died and valuable lessons were learned from each. Four of the cases were anesthetized with morphin followed by ether. The fifth one was anesthetized with nembutal. This one died under

*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

the anesthetic. Our experience with nembutal has led us to conclude that it is far inferior to morphin and ether as a general anesthetic for the dog. The fifth case died from hemorrhage due to opening a blood-vessel by scratching the operative wound. It is necessary to protect the wound for a week or ten days following the operation.

CASE 1

Subject: Male Springer Spaniel, owned by Z. K., Pullman, Washington.

History: This dog entered the hospital in November, 1929, with unilateral otitis externa. At this time it was a pup about two months old. The external ear was cleansed thoroughly with ether and treated with a solution of camphor and phenol. Nothing more was heard from this dog until July, 1930, or eight months later, when it was returned to the hospital with a profuse discharge of fetid, dark brown material from the same ear which had been treated before. The owner had poured "Glover's Mange Cure" in the canal, and this had produced such severe swelling and blisters as practically to occlude the passage. It was a week before the inflammation had subsided sufficiently to permit an examination to be made.

Symptoms: The dog frequently shook its head and carried the head slightly lower on the affected side. Considerable pain was experienced on palpation of the conchal cartilage, but very little on palpation of the bulla ossea. The bulla felt considerably larger than that on the normal side. After the external meatus had been cleaned sufficiently to allow passage of the otoscope, an apparently endless quantity of pus was encountered, making it evident that it was coming from the tympanic cavity. No tympanum was found.

Treatment: Bulla-osteotomy was performed. Upon the opening of the bulla, about two cubic centimeters of pus ran out of the external ear. The bone was greatly thickened and of a spongy nature. The wound was kept open for three weeks, when all evidence of a discharge had stopped.

This dog has had no recurrence of ear trouble of any kind during the past two years. The owner reports the dog in perfect health, but states the dog's hearing is not quite so acute as before the operation.

CASE 1425

Subject: Female Airedale Terrier, owned by a dentist in Oregon.

History: The owner reported the dog as having had a continuous discharge from the right ear for the past eighteen months. It was becoming particularly cross, not allowing even the owner to touch its head.

Symptoms: The dog seemed greatly depressed and held its head very low, but not to the side. An examination under a general anesthetic revealed a dark brown, thick, fetid discharge from the external ear and the deeper part of the meatus filled with pus. The bulla ossea was enlarged on the right side.

Treatment: Bulla-osteotomy was performed, September 20, 1930. The bone was thickened and spongy. The cavity of the bulla filled with pus, which did not run out of the external ear as in the previous case, due to the fact that the tympanum was more nearly complete. The case was discharged from the hospital, October 12.

On March 19, 1931, the owner responded to an inquiry in part as follows:

> The dog is just fine, and as far as I am qualified to judge, I would say your operation on her was a complete success. The dog's hearing is even better now than for some time before you operated on her. The carriage of the head is now normal.
>
> There has been much less trouble with constipation, no doubt due to the fact that there was a constant absorption into the system from the infected area.

CASE 3

Subject: Male Cocker Spaniel, owned by E. J., Colfax, Washington.

History: This dog had been ailing but a short time; at least the owner had observed symptoms not more than three weeks.

Symptoms: The patient held its head to the right side, shook it frequently and scratched the ear. A copious sanguinous discharge came from the affected ear, but did not have the extreme odor noticed in the more chronic cases. An examination of the ear revealed the canal full of fluid pus that bubbled up when the dog breathed deeply or howled. The deeper part of the canal was partially filled with a gray pus and white flaky material. The tympanum was very red and inflamed. While no opening could be seen in the drum, it apparently was not whole, as bubbles came through during deep respirations. Both the external acoustic meatus and the bulla ossea were very sensitive.

Treatment: Bulla-osteotomy was performed, February 24, 1931. A very small amount of bloody pus was found in the tympanic cavity. The mucous membrane was highly congested and inflamed. A light through both the external meatus and the

opening in the bulla revealed a small opening through the tympanum. Six months after the operation this dog was reported in perfect health.

Case 1879

Subject: Airedale Terrier, eight years old, owned by R. T. M., Garfield, Washington.

History: Both ears discharged for two years. The ears would appear to heal in hot weather and break out again in cold weather. The owner wished something done for the dog because of the extremely bad odor.

Symptoms: The head was carried low. The animal would frequently shake the head and then scratch at one ear and then the other. Both ears were so tender they could not be examined without anesthesia. Both ear drums were ruptured. The bulla ossea were about the same size, but probably larger than normal.

Treatment: Under nembutal anesthesia, bulla-osteotomy was performed, February 20, 1931. The tissues seemed very thick and heavy. Blunt dissection was very difficult and the tissues were hard to hold in the required position. The operation was completed, revealing pus in each bulla, and perfect drainage established. This animal never came out of the anesthesia.

Case 2264

Subject: Male Collie, eleven years old, owned by H. M. W., Moscow, Idaho.

History: This dog had ear trouble for a long time. He carried his head to one side and shook it frequently. Owner reported dog having a very bad odor. The old fellow was getting very cross the last year or so and could hear but very little. He would bite his owner if the latter touched his head.

Symptoms: Examination was carried out only under a general anesthesia. The external ears on both sides were filled with pus. One drum was perforated and the other one whole.

Treatment: Double bulla-osteotomy was performed, May 16, 1931. This dog had a very thick, heavy neck, hard to operate, which is characteristic of old dogs. The patient did fine for four days, eating and drinking well. On the night of the fourth day, the dog scratched open one of the blood-vessels on the left side, and died about 36 hours later, after repeated hemorrhages, which defied all surgical efforts to check.

Reference

[1]McNutt, G. W., and McCoy, J. E.: Bulla-osteotomy in the dog. Jour. A. V. M. A., lxxvii (1930), n. s. 30 (5), pp. 617-628.

BLACK TONGUE*

By H. CALVIN REA, *Charlotte, N. C.*

HISTORY

In 1850, Hoffer described a disease of dogs under the name "typhus." This disease created general interest when it appeared in 1898, in Germany and other countries, as a destructive plague. This outbreak was described first by Klett, who observed it in and around Stuttgart; hence the name "Stuttgart dog disease." This disease is said to have been observed first in America in 1887.[1] The description of the condition in some respects agrees with that of what is known today as "black tongue," so prevalent among dogs of the southern states. Very little more was written regarding this disease until 1911. Quoting from a historical review in *Veterinary Medicine*, August, 1928:

> Edgar Heiny reported black tongue as not infectious, October, 1911. L. F. Koonce, of Raleigh, N. C., December, 1911, reported the disease as not communicable; one attack gives no immunity to further attacks; raw meats constitute an effective treatment; inoculation experiments were negative. Dr. Koonce does not say in so many words that black tongue is a dietary disease but it is reasonably inferred from his report that he so regarded it.
>
> John A. Davis, March, 1912, stresses stiffness as a symptom and mentioned the use of diphtheria antitoxin (an animal product) as being curative. John M. Keer, Paris, Texas, December, 1914, pointed out that black tongue is identical to European canine typhus. T. N. Spencer, of Concord, N. C., April, 1916, was positive that black tongue in dogs is identical to pellagra in man and reported having cured every case treated, with milk, eggs and raw meat. The inference that the disease is dietetic and not infectious is unmistakable.
>
> G. W. Browning, February, 1917, states positively that black tongue is not infectious and that it may be cured with milk and beef broth. C. G. Stallworth, May, 1923, recommends forced feeding with salmon, broth and raw meat.

In 1915, Goldberger and associates, in beginning their studies of pellagra, after reading "The Nutrition of Man," by Chittenden, decided to use the dog as an experiment animal. In 1917, Chittenden and Underhill published a report on "the production in dogs of a pathological condition which closely resembles human pellagra," by means of a diet of boiled peas, cracker meal and cottonseed oil.[2]

Except for an interruption during the World War, Goldberger, Wheeler and associates did extensive investigational work with black tongue and pellagra, from 1915 to 1930 inclusive. Various Public Health Service reports give details of their studies. Dr.

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*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

T. W. Kemmerer, Director of State Hygienic Laboratory, Jackson, Miss., made a cultural study of the heart-blood of four cases at necropsy. His reports were negative. Experimental attempts at artificial transmission, with one or two doubtful exceptions, failed. Most of their work, however, was done along dietary lines.

Occurrence

Black tongue is much more prevalent in the southern states, if not entirely confined therein. Although a greater number of old dogs become affected, puppies are not immune. The dog is the only known susceptible domesticated animal. Ninety-three per cent of the patients were treated from March 1 to December 1.

Opinions Expressed by Other Writers

Müller and Glass, Hutyra and Marek, and Brumley use the following terms synonymously: infectious hemorrhagic gastroenteritis, canine typhus, Stuttgart dog disease, dog plague and black tongue.

Goldberger, Wheeler, Blackstock and associates express the opinion that there is a definite relationship between black tongue of dogs and pellagra of man. Their experimental evidence indicated that both diseases were caused by the lack of a single vitamin, recently termed vitamin B or G. They claim to have produced black tongue (pellagra) by giving a diet deficient in this vitamin and to have cured *either* of these conditions by giving a diet rich in it, namely, brewer's yeast, fresh raw beef, canned salmon and liver extract. [3-5]

Current veterinary literature does not give any detailed studies on the subject of black tongue by veterinarians. They apparently have accepted the deficiency theory without endeavoring to prove or disprove it.

Personal Experiences

Since 1918, the writer has observed that the percentage of cases of black tongue has remained relatively the same as compared with other diseases of canine patients.

Artificial transmission has been attempted by animal-to-animal contact, subcutaneous and intraperitoneal injection of blood, oral administration of saliva and the feeding of feces and urine; all attempts were unsuccessful. Fecal examination for parasites in 73 black tongue patients were compared with an equal number of fecal examinations of other hospital patients that were of approximately the same age. The results do not indicate a definite relationship between intestinal parasitism and black tongue.

Apparently it is not communicable; however, the possibility of transmission by parasites or insects has not been positively eliminated.

An attempt was made to ascertain the percentage of patients that recover without the services of a veterinarian. Two hundred and fifty-six persons, of at least average intelligence, were interviewed. Each person claimed to have owned one or more dogs afflicted with black tongue. These owners were questioned regarding the symptoms and only those are included that were probably diagnosed correctly. The results indicate that 15 per cent of unmedicated patients recover and that 4 per cent of the patients treated by quacks, with patent medicines and home remedies, recover.

Influence of Breed, Sex and Age

Hounds, pointers, setters and collies from suburban and rural districts constitute 85 per cent of the patients treated.

Males and females have been treated in equal number. The ages range from four months to twelve years; the average is three and one-half years. A review of the diet histories of patients indicates the following:

Scraps from the table.....42 per cent
Commercial dog feeds.....27 per cent
Beef or salmon at least twice a week.....17 per cent
General mixture of table scraps, beef and commercial dog feeds .....14 per cent

Not one patient has come from a home where there was a known pellagrin.

Case I—*Case reports, with treatment.*

Treatment	Cases	Recoveries	Mortality Rate (%)
Brewer's yeast	67	48	30
Sodium chlorid and glucose	46	39	15+
Lime water and calcium gluconate	42	30	29
Carotin extract in glycerin	9	6	33+
Liver extract	18	9	50
Liver extract and sodium chlorid	32	28	13—
Totals	214	160	Avg. 25+
Sodium chlorid	241	207	14+

These were all hospital patients and were treated from June 1, 1928, to August 15, 1932. During the 18 months prior to this

time, brewer's yeast was given all patients and the mortality rate was 30 per cent.

In an attempt to determine the relative value of various therapeutic agents, the following method was employed: When patients were admitted to the hospital, each one was numbered in regular order. With few exceptions, saline solution was given to those with even numbers, and those with odd numbers received the other agents indicated in table I.

As a mouth wash a solution of neutral acriflavine, potassium permanganate, or metaphen has been used to advantage.

Gastro-intestinal lavage is indicated, provided the patient is not too weak and does not offer a great deal of resistance. The solutions used are physiological saline or 2 per cent solution of sodium bicarbonate. Stimulants such as atropin sulfate, caffein and camphor apparently are valueless.

Cases are of two types—those with and those without nervous symptoms—but both types exhibit gastro-intestinal, symptoms with skin lesions.

Symptoms

The onset is sudden, with refusal of food or vomiting, anemia and emaciation, dehydration, depression, injected conjunctivae, eyes sunken, staggering gait and increased thirst. The buccal mucosa may be slightly reddened or it may show a marked necrosis which first appears inside the upper lips and cheeks and on the floor of the mouth. The tongue at no time becomes black. Temperature is 103 to 106° F., later dropping to subnormal. The pulse is rapid and weak, and respiration is accelerated and apparently painful. The urine is bile-stained and has an offensive odor. There is drooling of ropy saliva stained with blood in well developed cases, soiling the front legs. A very characteristic foul odor is emitted. Erythematous patches may be noted in various places. Occasionally a shifting lameness is present.

At the onset, most patients are constipated but lated develop a profuse bloody diarrhea. Nervous symptoms which manifest themselves by convulsions of the clonic type are occasionally noted; these invariably prove fatal.

Laboratory Findings

A chemical study of the blood and urine in 17 severe cases of black tongue was made. The blood showed an increase in nonprotein nitrogen, urea nitrogen, uric acid and carbon dioxid combining power. Assuming that 468 mg of blood chlorids (as NaCl) per 100 cc of blood is normal, the chlorids were reduced

from 15 to 50 per cent. The urine was high in nitrogen, low in chlorids and contained albumin and casts.

Microscopic and cultural studies of the blood, urine, feces, saliva and skin failed to associate a microörganism with this disease.

Differential Diagnosis

In our opinion, the above-mentioned authors have erred in an attempt to make Stuttgart disease, canine typhus, infectious hemorrhagic gastro-enteritis and black tongue one and the same disease.

Canine typhus is a term used synonymously with Stuttgart disease by present-day writers and these two terms are used interchangeably by many clinicians also. Because of its appropriateness, we prefer the term acute gastro-intestinal toxemia. It is different from black tongue in that it is an intoxication of digestive origin. The exact cause is unknown. There is early excessive thirst and vomition, due to an irritated stomach which becomes inflamed after intestinal lesions are well established. An almost constant symptom is the excessive dilation of one or more arteries, with or without visible branches, in the sclerae. The buccal inflammation is not confusing after a short period of observation. The mucosa shows reddened, restricted, ulcerated patches which do not slough or become necrotic as in black tongue. These patients do not emit the characteristic odor. If drooling occurs, it is more watery and not blood-stained. It is more acute. It kills by intoxication, dehydration and depression deepening into coma.

Stomatitis from such causes as hot feed, irritating chemicals or mechanical injuries should not be confusing.

Pathology

Severe inflammatory changes occur in the entire digestive tract. There is extensive necrosis of the buccal mucosa; the tongue is swollen and very red; the stomach is reduced in size and its mucosa greatly swollen; the small intestine is ulcerated and presents numerous hemorrhagic areas; the peritoneum over the stomach and intestines is injected; the mesenteric lymph-glands are enlarged and show acute swelling; the spleen, liver and kidneys are hyperemic, and the lungs edematous.

Course and Prognosis

Ninety-seven per cent of deaths occur within the first four days after entering the hospital. Assuming that the histories

given are correct, the average patient enters the hospital on the fifth day after the onset; thus death occurs on about the ninth day. Patients manifesting extensive nervous involvement invariably die. The patients received an average of ten days of hospitalization. One patient is known to have survived a fifth annual attack, five had three attacks (two of these died), and seven had two attacks. Dogs affected with this disease either die or recover completely.

Treatment

Nursing alone will not save black tongue patients, but all other treatment is futile if this important factor is neglected. General hygienic methods must be rigidly employed. Exposure to direct rays of the sun when the temperature is 80° F., or above, will invariably prove fatal after a short time.

Diet is unquestionably an influential factor in causing black tongue and it is equally as important in the treatment. The following foods have proved to be the most valuable: fresh raw beef muscle and canned salmon. An animal may be coaxed to eat and even aided in getting food in the mouth, but forced feeding is contraindicated.

Conclusions

1. Black tongue is a disease peculiar to the dog and is most prevalent in the southern states.

2. Veterinarians were first to express the opinion that it is a dietary deficiency disease. This was in 1911.

3. Goldberger, Wheeler and associates have reported more investigational work than any others.

4. In our experience neither brewer's yeast, raw liver, milk, milk and egg yolk, milk and karo syrup, nor beef broth has any value.

5. A greater percentage of unmedicated cases result in recovery than do those in which treatment is given by quacks, or when it consists of patent medicines and home remedies.

6. Liver extract used in conjunction with sodium chlorid shows approximately 1 per cent, the lowest death rate; however, only 32 patients were given this combination. In 18 cases liver extract alone was given, nine of these resulting in death.

7. Best results in clinical experience must ever remain the final and crucial test of every form of therapy. As far as results in so small a series can be trusted, the symptoms manifested by black tongue patients are very definite indications for sodium chlorid, regardless of the cause. Depleted blood chlorids are

replaced, the fluid content of the body is increased, normal peristalsis is restored, the kidneys are flushed and metabolic processes are materially aided.

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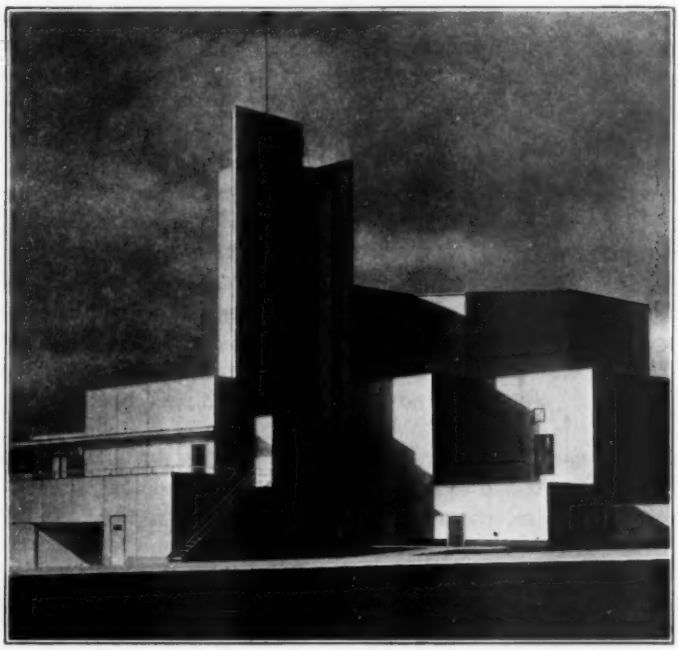
[3]Goldberger *et al*: Public Health Reports, xl (1925), pp. 54-80.
[4]Goldberger *et al*: Public Health Reports, xl (1925), 19, pp. 927-928.
[5]Wheeler, G. A.: Black tongue in dogs and its relation to pellagra in the human. Jour. A. V. M. A., lxxvii (1930), n. s. 30 (1), pp. 62-72.

BUREAU TRANSFERS

DR. L. S. SMITH (Colo. '18) from Trinidad, Colo., to South Saint Paul, Minn., on meat inspection.

DR. F. M. WELCH (McK. '18) from Saint Joseph, Mo., to Montgomery, Ala., on meat inspection.



DAIRY BUILDING AT A CENTURY OF PROGRESS

In this structure, of cubistically modified oval shape, will be portrayed the story of the foster mother of mankind, the cow. The evolution of dairying methods during the last one hundred years will be unfolded before the visitor's eye in a spectacular fashion. This will be the only building at Chicago's 1933 World's Fair, the exterior of which will be painted white.

A PRELIMINARY REPORT ON THE STUDY OF POULTRY VERMIFUGES*

By E. F. THOMAS, *Gainesville, Florida*
Florida Agricultural Experiment Station

Much uncertainty long has existed regarding the value of giving vermifuges to poultry. The conflicting results obtained from the use of poultry vermifuges, and the lack of experimental data regarding their value and effect on egg-production has made it evident that research work on such a problem was needed.

The recommendation usually made in Florida regarding worming chickens is to give all pullets and hens some kind of vermifuge for roundworms and tapeworms before putting them in the laying quarters. The writer has been recommending, for the past several years, tetrachlorethylene and kamala as described in U. S. Department of Agriculture bulletins 1337 and 1652, on diseases and parasites of poultry. In this State many poultry advisers are recommending this and other treatments. The one treatment probably used as much or more than the tetrachlorethylene-kamala treatment is "iodine vermicide." Since this was true it was decided to compare the value of these two widely used treatments and thereby determine, if possible, which treatment gave the better results, as measured by egg-production and general health of the flock.

In all tests, tetrachlorethylene and kamala were used as recommended by Schlingman.[1] A 1-cc capsule of tetrachlorethylene was given to each bird, followed in three days by a 1-gram compressed tablet of kamala.

In all tests iodine vermicide was administered and used as recommended by Chandler.[2] A 1-ounce dose of the mixture was administered directly into the gizzard with a flexible hard-rubber catheter.

The results of four experiments are included in this paper, the experiments being designated as A, B, C and D.

EXPERIMENT A

November 15, 1930, 75 S. C. White Leghorn pullets, six to seven months old, were selected from a flock that had been wormed once several weeks previously and, according to the owner, found to have numerous ascarids. Three birds from the flock were posted and showed a heavy infestation of tapeworms

*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

(*H. carioca*). These birds were allowed to stay on the same contaminated soil two weeks to assure reinfestation with ascarids. The 75 birds then were moved to the experiment pens and divided, three groups of 25 each to a pen. The birds were leg-banded and graded as evenly as possible. Feeding and management practices were the same for all three groups. During the last week of December, 1930, all three pens showed ascarids on the dropping-boards. January 1, 1931, trap-nesting was started. January 30, each of the 25 hens in pen 1 was given a 1-cc capsule of tetrachlorethylene, and on February 2, a 1-gram tablet of kamala. January 30, each of the 25 hens in pen 2 was given a 1-ounce dose of iodine vermicide. The 25 hens in pen 3, with no treatment, were kept as controls. January 31, 1931, numerous ascarids were found on dropping-boards of pens 1 and 2. Freshly voided ascarids were found on dropping-boards of both treated pens the second day following treatment. No tapeworms were observed in the droppings following the kamala. Apparently, no ill effects were suffered by the birds from either treatment. One bird in pen 1 that was thin in flesh and affected with leg paralysis withstood the treatment without apparent harm.

TABLE I—*Experiment A. Parasitic infestation of birds that died during six-month test-period.*

	Pen 1 (Tetra-chlor-ethylene-Kamala)		Pen 2 (Iodine Vermicide)		Pen 3 (Untreated)	
Month	Deaths	Parasites	Deaths	Parasites	Deaths	Parasites
January..	0	—	1	1-ascarids	1	1-ascarid 1-tapeworms
February.	2	No parasites	0	—	1	1-ascarid 1-tapeworms
March...	2	1-ascarids 1-tapeworms	3	3-ascarids 1-tapeworms	3	3-ascarids 1-tapeworms
April.....	4	4-ascarids 1-tapeworms	1	No parasites	3	1-ascarid 1-tapeworms
May.....	1	1-ascarids	2	2-ascarids	2	2-ascarids 1-tapeworms
June.....	2	2-capillarids 1-ascarids	6	4-ascarids 1-capillarids	6	2-ascarids 1-tapeworms

Table I shows that both ascarids and tapeworms were present again in birds from pens 1 and 2 in less than two months following treatment. This was to be expected, since the birds were placed in the experiment pens one month before being

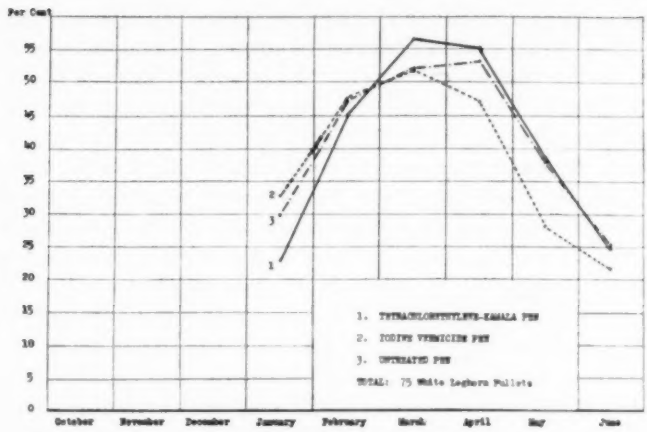


FIG. 1. Chart showing effect of worm treatment on egg-production (Experiment A).

treated. Ascarids and tapeworms were found in the untreated pen from January to July.

From a study of the chart shown in figure 1 it will be noted

TABLE II—*Experiment A. Effect of worm treatment on egg production as shown by daily egg-record (20 days taken from trap-nest record).*

DATE	TREATMENT	EGGS		
		PEN 1 (25 HENS)	PEN 2 (24 HENS)	PEN 3 (24 HENS)
January 29–31		8	11	13
January 30	Tetrachlorethylene (Pen 1) Iodine vermicide (Pen 2)	12	10	4
January 31		12	12	12
February 1		8	10	11
February 2	Kamala (Pen 1)	13	9	11
February 3		13	10	12
February 4		11	9	9
February 5		5	9	10
February 6		8	11	7
February 7		8	13	11
February 8		4	11	7
February 9		6	12	9
February 10		2	14	13
February 11		4	8	10
February 12		13	13	12
February 13		8	12	10
February 14		13	10	12
February 15		17	15	9
February 16		13	11	12
February 17		15	11	10
		(24 hens)	(23 hens)	(24 hens)

that the untreated pen was slightly above the average in egg-production from January through June, although pen 1 was ahead during March and April. As shown in table VII, the untreated pen had the highest average percentage production for six months, and this was likewise true through the remainder of the year. Average percentage production for 13 months for pen 1 was 26.26 per cent; pen 2, 21.91 per cent; and pen 3, 27.57 per cent. Pen 1 was highest in March and April but showed a steady drop from April to July. In table II the daily egg-record shows that pen 1 suffered a decrease in egg-production that lasted seven days, starting on the third day following the administration of kamala. This decrease seems to have been made up partially, since pen 1 came out with a 2.3 per cent lead over pen 2 for the six months. These results are very similar to the findings of Atwood and Clark.[3] Their data show that egg-production was not affected materially until the third day and lasted nine days following a 1-gram dose of kamala. Pen 3 shows a 14.17 per cent higher mortality for the six months over pen 1 and 16 per cent higher mortality than pen 2 (table VIII).

TABLE III—*Experiment A. Relation of feed consumption to number of eggs laid from January to July (6 months).*

PEN	TREATMENT	EGGS	FEED (LBS.)	FEED CONSUMED (LBS. PER DOZ.)
1	Tetrachlorethylene-kamala	1,636	739	5.42
2	Iodine vermicide	1,486	739	5.96
3	None (controls)	1,608	722	5.39

In table III it is shown that 0.58 pound more feed was required for birds in pen 2 and 0.03 pound more for pen 1 than in case of pen 3 (untreated) to produce one dozen eggs.

Table IV shows the relation between egg-production and worm infestation, the latter being based upon the number of ascarids expelled following treatment. This is only a relative index to the worm infestation, since it was impossible to destroy the birds for postmortem examination and determine the exact infestation.

Results obtained from these preliminary studies with 75 hens seemed to indicate that the worm treatment used in these studies did not improve the health of the bird or increase egg-produc-

TABLE IV—*Experiment A. The relation between egg-production and worm infestation.*

HEN	EGGS LAID DURING 189-DAY PERIOD	ASCARIDS PASSED	TREATMENT
916	137	77	Tetrachlorethylene
902	130	0	Tetrachlorethylene
868	121	64	Iodine vermicide
884	109	37	Iodine vermicide
930	60	90	Tetrachlorethylene
914	43	8	Tetrachlorethylene
883	32	2	Iodine vermicide
888	6	1	Iodine vermicide

tion. It was decided, therefore, to carry out similar experiments under commercial poultry farm flock conditions.

Three poultrymen were found who were willing to take a chance on an untreated pen after being shown the results obtained with experiment A. These poultrymen were planning on worming their flocks when the birds were put into the fall laying-houses.

EXPERIMENT B

Nine hundred and forty-seven S. C. White Leghorn pullets just coming into production were divided into three lots as follows: pen 1, 318 birds; pen 2, 314 birds; and pen 3, 315 birds. Birds in all pens were as uniform in all respects as it was possible to obtain. All birds were hatched in April and were of the same strain of Leghorns. Although the flock was not one that could have been considered as heavily infested, yet many birds showed symptoms of worm infestation. Several birds were light in flesh, and about one-third of the group showed scaly combs. A postmortem examination of two birds which showed these identical symptoms revealed that they were heavily infested with ascarids and tapeworms (*H. carioca*).

The pullets were placed in three separate houses (16′ x 48′) with concrete floors and "wired-in" dropping-boards. These houses were used the previous year, but for this experiment they were cleaned thoroughly and disinfected with a fire-torch. Ranges which had not been used by birds for at least one year were provided. Feeding and management practices were the same for all three pens.

Treatment consisting of tetrachlorethylene and kamala was given to pen 1, October 16 and 19, 1931, respectively. Pen 2 was

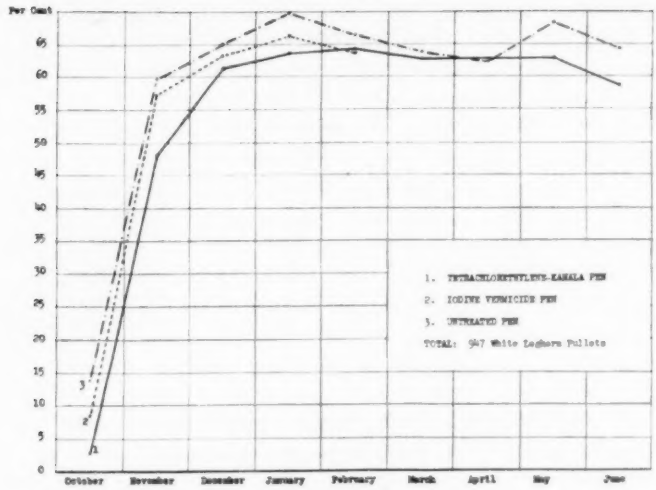


FIG. 2. Chart showing effect of worm treatment on egg-production (Experiment B).

given iodine vermicide, October 16, 1931. The birds in pen 3 were kept as controls and were not given treatment. All birds in the control pen, as well as the treated pens, were kept in the houses four days and the houses cleaned of droppings and litter at the termination of this period.

About one hour following administration of iodine vermicide, several birds in pen 2 expelled tapeworms (*T. infundibuliformis* and *D. tetragona*) in loose bowel movements. The owner reported that he observed only a few ascarids on the dropping-boards the following morning. The treatment was given about 10 o'clock in the morning.

The chart in figure 2 shows the egg-production of the three pens in experiment B. Pen 2 of this experiment was discontinued, February 29, due to the owner having sold the birds at an attractive price. A five-month record was obtained, however, on these birds. Table VII shows the average percentage production for nine months for pens 1 and 3, and five months for pen 2. Pen 3, containing the untreated birds, led in egg-production by a margin of 5.18 per cent over the next nearest pen, which was pen 1.

Table VIII shows the percentage mortality of pen 1 to be 3.40 per cent higher than pen 3, the untreated pen. Table VI shows that the untreated pen (3) required less feed (0.384 pound) than the treated pen (1) to produce a dozen eggs for the nine-month

period. Table V shows that the untreated pen required 0.444 pound less feed than the tetrachlorethylene-kamala pen, and 0.096 pound less feed than the iodine-vermicide pen to produce a dozen eggs for the five-month period when pen 2 was on test.

TABLE V—*Experiment B. Relation of feed consumption to number of eggs laid from October to March (5 months).*

PEN	TREATMENT	EGGS	FEED (LBS.)	FEED CONSUMED (LBS. PER DOZEN)	AVERAGE PRODUCTION (FOR 5 MONTHS) (%)
1	Tetrachlorethylene-kamala	21,894	7,966	4.368	48.02
2	Iodine vermicide	22,854	7,649	4.020	51.72
3	None (controls)	23,696	7,758	3.924	54.82

TABLE VI—*Experiment B. Relation of feed consumption to number of eggs laid from October to July (9 months).**

PEN	TREATMENT	EGGS	FEED (LBS.)	FEED CONSUMED (LBS. PER DOZEN)	AVERAGE PRODUCTION (FOR 9 MONTHS) (%)
1	Tetrachlorethylene-kamala	41,628	14,769	4.260	54.11
3	Untreated	44,430	14,336	3.876	59.29

*Pen 2 discontinued, March 1, 1932.

EXPERIMENT C

Two hundred and fifty S. C. White Leghorn hens were selected by the owner from his pullet flock of the previous year and divided into three lots as follows: pen 1, 100 hens; pen 2, 72 hens; and pen 3, 78 hens. Birds in all three pens were as nearly uniform in type as it was possible to select.

During the previous year, hens from this flock had been presented to our laboratory for postmortem examination and were found to be infested with ascarids and tapeworms. Scaly combs and wattles were evident in the flock and several showed gray eyes.

The hens were placed in three separate houses (12′ x 16′), each being provided with sawdust litter on the floors. This was

changed once a week. Droppings collected in the sawdust on the floor, since no dropping-boards were used. Separate runs were provided. These houses and runs had been used practically continuously by chickens for the past seven years. Feeding and management practices were the same for all three pens.

Treatment consisting of tetrachlorethylene and kamala was given to birds in pen 1, October 30 and November 2, 1931, respectively. Pen 2 was given iodine vermicide, October 30, 1931. A few ascarids were noticed in droppings passed by birds given iodine vermicide about one hour after treatment. The owner did not notice any worms in the droppings the morning after treatment. Since the birds had access to the droppings, such an observation, made a few hours after the birds were off their roosts, could not be conclusive.

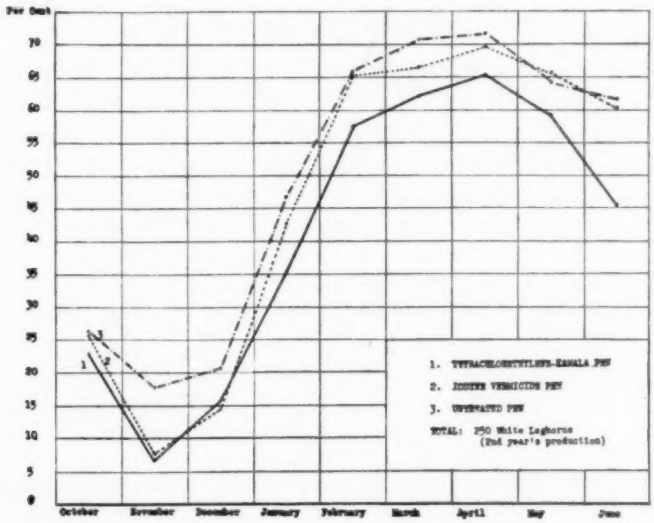


FIG. 3. Chart showing effect of worm treatment on egg-production (Experiment C).

The chart in figure 3 shows the percentage egg-production for nine months. The two treated pens both suffered a decreased egg-production following treatment and never regained production equal to that of the untreated pen. The untreated pen was first in egg-production, with the iodine-vermicide pen second. The untreated pen led pen 2 by a margin of 3.15 per cent for the nine-month average production, as is shown in table VII. Pen 3

(untreated) had a slightly higher (1.71 per cent) mortality than pen 2, and 6.82 per cent higher than pen 1, as shown in table VIII. No feed records were kept on this flock.

TABLE VII—*Effect of worm treatment as shown by the average percentage production of all pens of each experiment.*

EXPERIMENT	MONTHS REPRESENTED	BIRDS AT START OF EXPERIMENT	PEN 1 (TETRACHLORETHYLENE-KAMALA) (%)	PEN 2 (IODINE VERMICIDE) (%)	PEN 3 (UNTREATED) (%)
A	6	75	40.52	38.17	40.85
B	9	947	54.11	51.72*	59.29
C	9	250	41.13	46.38	49.53
D	9	357	36.36	34.03	41.17

*Five months. Pen 2 discontinued, March 1, 1932.

TABLE VIII—*A comparison of average percentage mortality of treated and untreated pens of each experiment.*

EXPERIMENT	MONTHS REPRESENTED	BIRDS AT START OF EXPERIMENT	MORTALITY (%)		
			PEN 1 (TETRACHLORETHYLENE-KAMALA)	PEN 2 (IODINE VERMICIDE)	PEN 3 (UNTREATED)
A	6	75	40.00	36.00	54.17
B	9	947	10.38	4.46*	6.98
C	9	250	6.00	11.11	12.82
D	9	357	8.21	9.46	7.29

*Five months. Pen 2 discontinued, March 1, 1932.

EXPERIMENT D

Three hundred and fifty-seven S. C. White Leghorn hens were selected by the owner from his flock of hens that had finished their second year of egg-production, and divided into three lots as follows: pen 1, 134 hens; pen 2, 127 hens; and pen 3, 96 hens. The three lots of birds were as nearly uniform as they could be selected. This flock was at a very low level of egg-production and numerous birds had scaly combs and wattles. Ascarid ova

had been found in a fecal sample examined by the writer September 24, 1931.

The hens were placed in three separate houses (10′ x 24′) having concrete floors but no dropping-boards. All birds used the same common range. This range or yard had been used by chickens continuously for the past four years. Feeding and management practices were the same for all three pens.

Treatment was administered, as was described in experiments A, B, and C, to pen 1, tetrachlorethylene and kamala, November 11 and 14, 1931, respectively. Pen 2 was given iodine vermicide, November 11, 1931. The owner reported that he noticed no worms expelled by the birds treated.

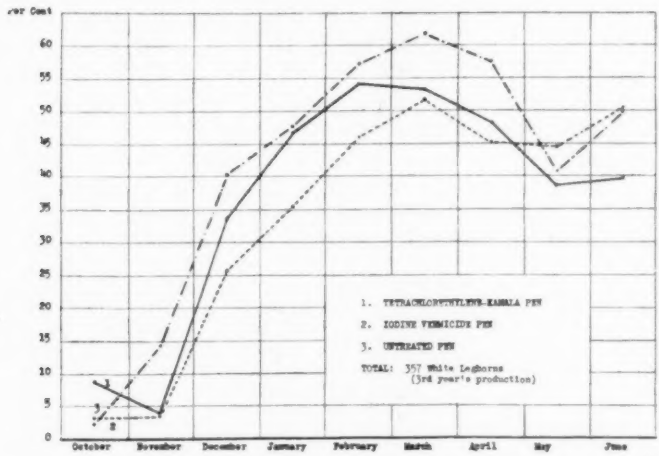


FIG. 4. Chart showing effect of worm treatment on egg-production, 1931-32 (Experiment D).

The chart in figure 4 shows the percentage egg-production for nine months. The tetrachlorethylene-kamala pen suffered a decreased egg-production following treatment. The untreated pen was first in egg-production, with the tetrachlorethylene-kamala pen second. Pen 3 (untreated) led pen 1 in egg-production by a margin of 4.81 per cent, and pen 2 by 7.14 per cent, as is shown in table VII. Pen 2 (iodine vermicide) had slightly the highest percentage mortality, 1.25 per cent higher than pen 1 and 2.17 per cent higher than the untreated pen. (See table VIII.)

Table IX shows the parasitic infestation at the termination of experiments B, C and D, determined by postmortem examination of two birds from each pen. All three field experiments showed

ascarid infestation at their termination. Experiments C and D showed tapeworm infestations.

TABLE IX—*Parasitic infestation at termination of experiments B, C and D, as shown by postmortem examination of two birds taken from each pen. (Field experiments.)*

EXPERIMENT	PEN	BIRD	CONDITION	POSTMORTEM FINDINGS
B	1	1	Apparently healthy (cull)	No parasites
B	1	2	Apparently healthy (laying)	Ascarids
B	3	1	Apparently healthy (cull)	No parasites
B	3	2	Apparently healthy (laying)	No parasites
C	1	1	Apparently healthy	Intestine filled with ascarids
C	1	2	Apparently healthy	No parasites
C	2	1	Apparently healthy	Ascarids
C	2	2	Molting	Ascarids and tapeworms (*D. cesticillus*)
C	3	1	Apparently healthy (laying)	Ascarids
C	3	2	Apparently healthy	Tapeworms (*D. cesticillus*)
D	1	1	Apparently healthy	Ascarids
D	1	2	Apparently healthy	Ascarids and tapeworms (*T. infundibuliformis*)
D	2	1	Apparently healthy	Ascarids
D	2	2	Apparently healthy	No parasites
D	3	1	Apparently healthy	Ascarids
D	3	2	Apparently healthy	Ascarids and tapeworms (*T. infundibuliformis*)

DISCUSSION

The results of the four experiments, with 1,629 birds of different ages and under different farm conditions, coincide with the findings of Bleecker and Smith,[4] in that the egg-production of their untreated birds was 1.50 per cent higher than the treated. They used only a small number of birds (average, 24.5) and administered five treatments to the treated pen.

The untreated hens in all four of the experiments finished the tests with higher percentage production, and in those experiments in which feed records were kept the hens consumed less feed per dozen eggs than the treated pens. The charts of percentage production show the curve to be of the same general trend in all the experiments with the untreated pen leading in production.

According to these tests, the tetrachlorethylene-kamala-treated pens were second in egg-production in experiments A and D, while the iodine-vermicide-treated pens were second in experiments C and B.

There seemed to be very little relationship, if any, between the mortality of the treated and untreated pens. The untreated pens showed the highest average mortality in experiments A and C. Mortality following the use of tetrachlorethylene-kamala was highest in experiment B, and of iodine vermicide in experiment D.

From the postmortem records of birds that died in experiment A, it appears that treatment probably removed the larger percentage of the worms, as none were found in two hens from pen 1 in February, while one hen from pen 3 showed both ascarids and tapeworms. Apparently reinfestation occurred in the treated birds very soon after the administration of the vermifuges, as is shown by postmortem examination of five birds from the treated pens 1 and 2. This may account for the treated birds being unable to regain the level in egg-production maintained by the untreated birds.

A decrease in egg-production is very evident in experiment C following treatment and in the tetrachlorethylene-kamala pen of experiments A and D. The pullet flock in experiment B was treated at the time production started. Therefore, this decrease could not be observed, but the treated pens showed a lowered production and never reached the level maintained by the untreated pen.

There seems to be no correlation between the number of ascarids passed and egg-production in infested hens, since the best layer passed 77 ascarids and the poorest layer only one, following administration of vermifuge.

All flocks on test were found to be infested before and after treatment. Where sanitary conditions were best, infestations appeared to be lightest.

The average number of birds on the experiment was 1,447.5.

Conclusions

1. These experiments indicate that hens given one worm treatment and placed back on the same ground are handicapped, as they suffer a decreased egg-production due to treatment, and are reinfested with ascarids and tapeworms before they have time to recover.

2. The evidence secured from these experiments shows that

worm treatment as practiced in Florida is apparently not only worthless but harmful and expensive.

3. There was very little indication of any superiority of one treatment over the other.

4. There was no correlation between the degree of ascarid infestation, as measured by evacuation due to treatment, and egg-production in laying hens.

5. It would seem, from the indications shown in these preliminary studies of the value of vermifuges, that the best procedure for poultrymen to follow would be to inaugurate sanitary measures to prevent infestation as far as possible. When infestation does occur, cull vigorously, and forget worm treatment until some vermifuge and method of use has been found, tested, and proven beneficial, by the use of control flocks. Poultrymen should not put confidence and faith in testimonials regarding the value of vermifuges, as has been done in the past.

Acknowledgments

The writer wishes to express his appreciation and thanks to Dr. A. L. Shealy, head of the Animal Husbandry Department; N. R. Mehrhof, Extension Poultryman; A. P. Spencer, vice-director of the Extension Division; Henry Smith, poultryman, Hilliard, Fla.; E. J. Eckenfels, poultryman, Green Cove Springs, Fla., and W. A. Ripton, poultryman, Mannville, Fla., for their courtesy and hearty coöperation which has been invaluable in these studies.

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Discussion

Dr. M. W. Emmel: When do you think a flock should be wormed?

Dr. Thomas: Well, I really do not know, but it would seem to me, from the work we have done, that if we are going to worm birds and get any beneficial effect from it, it should be done before they are three or four months of age.

Dr. Emmel: We have this condition in Alabama. We can control roundworms by sanitation but do not have much success in trying to control tapeworms in this manner, although we can control them to some extent. What are we going to do if we treat a flock to eliminate tapeworms, and in three or four months the chickens are infested again? Would you advise treating them again at that time?

Dr. Thomas: No, I would not.

DR. EMMEL: Would you advise treating them before they go into production, and then not treat them any more until after they had left the premises? Is that the idea?

DR. THOMAS: We have treated birds just before going into production, but our results indicate that it was unsatisfactory. We did not get good results from the treatment at that time; so I would hesitate to recommend worm treatment in any bird after it is four months of age.

DR. R. E. WOOD: What would be your advice as to sanitation? Did you rotate these flocks and leave the ground lying idle?

DR. THOMAS: We did not rotate these flocks. We do have poultry flocks in Florida that are rotated. These flocks have three ranges, and they are rotated so that they use their range only once every three years, and those poultrymen are controlling ascarids. I suppose that they have the same trouble which Dr. Emmel spoke of in dealing with tapeworms. They seem to control the tapeworm infestation to a certain extent, but our trouble is that we do not seem to be able to locate the intermediate host. They start their chicks in a sanitary brooder and then carry them on a range that has not been used for three years. They keep them on this range until they start laying, at which time they are transferred to the laying house, which has been disinfected and which has wired-in dropping-boards. The laying house range has not been used for one year. We are advocating this because our experiments have shown that ascarid ova disappeared from the soil in six months, six inches below the surface. Those on top of the ground surface disappeared in three months. For these reasons we recommend such a program. We have not carried these experiments as far as we hope to carry them.

DR. EMMEL: In Alabama I have always regarded parasites as being one of our chief problems. It has been my pleasure to be associated with poultrymen, particularly from the North, for years. They also have parasite problems, and it seems to me that we could gain some advantage by comparing our problems with theirs. We have them all the year round. That is true not only in poultry, but in other animals as well. We, in Alabama, many times meet other conditions which are, or which we feel are, secondary to the different infestations. Now Dr. Thomas said that, according to his experiments, birds should be treated before they are four months old. As a general rule we find very few birds under four months of age infested with worms to a point where we feel very much damage is done. Most of our parasitic infestations in poultry come after that. We sometimes have a somewhat chronic condition in adult birds. Do you have this, a chronic parasitic condition? I would also like to ask Dr. Thomas if, under those conditions, he would advise treatment of the birds for parasites?

DR. THOMAS: I hardly know how to answer your question since I have been working with this for only two years, and there are many questions which I am unable to answer. I do not know how to go about handling such a proposition; but it seems that if we could prevent infestation, we would get much better results than we would if we treated the birds after they are infested. I know that it is almost impossible to tell a poultryman not to do something with a bird in this condition, but from our experience in the field and the results obtained I would not advise such treatment.

DR. D. C. GILLES: In Florida we have many young birds which become infested before they are four months old, sometimes at six weeks of age. Even before that roundworms and tapeworms are seen occasionally. It seems to me that the best thing for us to do is to prevent worm infestation before it develops, if possible.

DR. WOOD: There is one thing that you have that we do not have. Dr. Emmel spoke of having trouble the year round. In my section of the country we figure that winter kills the worms to a certain extent. Our trouble with parasites comes along in the late fall, in October. Our pullets, a great many times, are wormed in early fall. I do not remember in my practice that I have ever been called to a flock of birds under six months of age. We generally figure that these late pullets which are to be put in the laying house for the winter are infested, because generally they are hatched about the latter part of March or the first of April. They become infested after they go into the laying-house, and we generally give them a round of kamala. We get pretty good results by using that for the roundworms. As for tapeworms, we are up against it; we do not know how to handle them.

DR. THOMAS: I might say that the same practice is true in Florida which may be due to most of our poultrymen being from the northern states. We have very few poultrymen who worm their pullets in the early stages. They worm them when they put them in the laying-house and worm the older birds at the same time.

DR. WOOD: One other thing—you have a longer period for the pullets to range than we have. Many times our pullets are put in the laying-house in September when they were not hatched until April.

DR. B. T. SIMMS: I want to compliment Dr. Thomas on his paper. I am very much interested in veterinary economics research work. We put birds in pens and treat them with the different kinds of vermifuges and then confer as to the advisability of using this or that treatment, but the farmer is not interested in how many worms are killed in a chicken. He is interested in how many chickens lay afterwards. He does not care about worms. All that he wants to know is the results. The work of Dr. Thomas, however, will be of great advantage to the farmer, whether he appreciates it or not. All the farmer wants to know is whether the chickens will lay afterwards.

New Bulletin on Equine Encephalomyelitis

Dr. Edward Records advises that the Nevada Agricultural Experiment Station will soon have available for distribution Bulletin 132, "Equine Encephalomyelitis," prepared by the staff of the Department of Veterinary Science of the Station, in collaboration with Dr. F. H. Baker, of Gardnerville, Nev., and Dr. E. T. Woodward, of Fallon, Nev., practicing veterinarians who have had extensive experience with this disease.

The bulletin will be semi-technical and is intended primarily for veterinarians, particularly those located in areas where the disease is apt to appear or recur this year in epizoötic form. The use of the recently developed antiserum, both for prophylactic and curative purposes, is discussed in the bulletin.

Any veterinarian whose name is not on the mailing list of the Nevada Agricultural Experiment Station may secure a copy of the bulletin by writing to Dr. Edward Records, University of Nevada, Reno, Nev.

FURTHER STUDIES ON THE RELATIVE EFFICIENCY OF VERMIFUGES FOR POULTRY*

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This study was inaugurated with the idea of determining the efficiency of a number of the most commonly used commercial vermifuges on the market, by comparing the efficiency of each with the others tested and, if possible, thereby placing us in a better position to advise others concerning the choice of agents for the removal of parasites from poultry.

There is no doubt in the minds of the writers that parasites—internal parasites particularly—are the one most important factor in determining the success of poultry-raising, in Arkansas at least. Out of 170 birds autopsied for Arkansas poultrymen during the fiscal year 1931-1932, fully 90 per cent were found to harbor one or more species of internal parasites and in 58 per cent of these cases the presence of parasites was the only appreciable cause of the unthriftiness, paralysis, or death. These results agree rather closely with those reported by Dearstyne and associates,[1] of North Carolina. They say:

> Intestinal parasites of the domestic fowl are responsible for an enormous annual loss in North Carolina, both directly and indirectly. The direct damage takes place through mortality, chiefly from the pullet age on, and loss in egg-production. The indirect loss is incurred by the fact that the mechanical injury brought about and toxins produced by the parasites predispose the fowl to other diseases.

Dearstyne[1] found the several parasites to be distributed as follows: 24.8 per cent of the fowls examined harbored *Ascaris lineata;* 13.8 per cent, tapeworms; 4.7 per cent, *Spiroptera hamulosa* (gizzard worms) and practically 100 per cent were infested with *Heterakis papillosa.*

Niemann,[2] in summarizing the results of necropsies of 93 paralyzed birds, reported that 45.16 per cent harbored tapeworms, 29.03 per cent harbored *Ascaris lineata* and 59.14 per cent harbored *Heterakis papillosa.*

Dearstyne also found a tendency for a seasonal incidence of mortality due to intestinal parasites. He reported losses less than 5 per cent in the first six months of the year and in December, over 10 per cent during the months of July and November,

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15 plus per cent in August and October, and more than 20 per cent during September.

While he gives no definite figures, Beach[3] says this relative to the damage done by intestinal parasites:

> During the last few years the Department of Veterinary Science of the University of Wisconsin has received hundreds of fowls for examination. The majority of these have been found to be infested with intestinal worms.

In discussing their influence on the host he says:

> Disregarding all doubtful cases there was left a large number of birds in which it was clear that the worms were responsible for the sickness. Most of the diseases of birds between three and ten months of age are either directly or indirectly caused by worm infestation.

Stafseth and Weisner[4] report relatively heavy losses due to intestinal parasites. In the eight years of the Michigan egg-laying contest, which started on November 1, 1922, 1,898 birds died and were autopsied. Of these, 114 (6 per cent) died because of infestation with intestinal parasites. The total mortality amounted to 18.98 per cent of the birds in the contest. These figures are particularly significant when we must assume that these birds are kept under the best possible sanitary conditions.

At the same institution,[5] during the years 1927, 1928, 1929 and 1930, 10,831 birds were sent in to the College for autopsy. Of these, 1,175 (10.8 per cent) were found to have died from the effects of intestinal parasites: tapeworms, roundworms and capillariae.

During the previous year's work it was found:

1. That there is a considerable difference in the efficiency of the commercial worm remedies studied. The one found by us to be the most efficient was the one prepared according to the formula of Dr. Stanley B. Freeborn,[6] of the University of California, when combined with fifteen grains of kamala. This vermifuge is prepared by the Eli Lilly Company, under the name "Pulvules No. 142," and consists of nicotin sulfate plus Lloyd's reagent.

2. That one treatment, with the vermifuge we found to be second in efficiency, was not sufficient to expel all parasites from heavily infested birds and it was concluded that with this vermifuge two administrations were necessary.

3. That repeated treatments, with this same vermifuge, of production pullets ranging on infested soil was of no advantage in increasing egg-production or in improving the health or vigor of the birds.

The experimental work during this second year was conducted

on the same general lines as in the first. As far as possible birds were purchased from flocks that had been found to be parasitized by the making of postmortem examination for Arkansas poultrymen. It was found, however, that not sufficient birds could be purchased from such flocks to meet our requirements, so a relatively large number of cull birds were obtained from one of the poultry produce houses. These were not so satisfactory as ones purchased from parasitized flocks. However, a large proportion were found to harbor intestinal parasites.

During this year, the study of vermifuges was confined to comparing the efficiency of but three preparations: nicotin sulfate, in the form of "Black Leaf 40," plus 15 grains of kamala; nicotin sulfate, as combined in Pulvules No. 142 (Lilly), plus 15 grains of kamala; and Iodine Vermicide (Merck), prepared according to the formula of W. L. Chandler.[7] With the exception of one group (8), each vermifuge was followed in three to four hours with one teaspoonful of Epsom salt. The salt was dissolved in water and administered to each individual bird by means of a funnel fitted with a soft rubber tube. The group consisting of twelve birds that did not receive the Epsom salt were given 0.4 grain of calomel.

Dr. Freeborn stated that nicotin sulfate in the form of Black Leaf 40 was too toxic for birds to be used alone, so combined it with fullers' earth with favorable results and in Pulvules No. 142 (Lilly), which are prepared according to his formula, it is combined with Lloyd's alkaloidal reagent. In the work herein reported, nicotin sulfate in the form of Black Leaf 40 was administered in amounts up to 0.3 cc in a gelatin capsule without producing any toxic effect. When used in amounts of 0.5 to 1.0 cc, a toxic effect was produced in badly depressed, unthrifty birds, and seven birds died in from two to ten hours following its administration. When used in from 0.4 to 1.0 cc, this preparation did not appear to be so effective in the removal of intestinal round-worms as when used in smaller amounts. In 30 birds, when used in varying amounts, up to a maximum of 0.3 cc, no toxic symptoms were produced and it was 100 per cent effective in removing *Ascaris lineata* from all birds harboring the parasite.

At the 1931 meeting of the United States Live Stock Sanitary Association, Dr. H. J. Stafseth,[8] of Michigan State College, presented a paper in which he reported very successful and effective use of iodine vermicide. It seemed desirable, therefore, to test this preparation and to compare its efficiency with that of Pulvules No. 142 (Lilly), the preparation that was found to be the most effective the previous year.

The writers were somewhat apprehensive relative to the practicability of the use of iodine vermicide. It is administered directly into the gizzard by means of a bulb-syringe, fitted with a long, slender, flexible nozzle, about eighteen inches in length. It was found, however, that there were no grounds for this fear as no difficulty was experienced in its proper administration.

Before tests were made of iodine vermicide under controlled conditions, a White Leghorn hen that had been sent to the laboratory for postmortem examination was given one ounce by means of the regular applicator. In less than thirty minutes she passed fully a tablespoonful of large tapeworms.

Table I gives a summary of the results obtained in the comparison of the three vermifuges. It does not give the detailed results of each bird autopsied, but a summary of the results obtained upon the autopsy of those in each group.

Where a vermifuge is listed as being 100 per cent efficient, this means that every parasite, with the exception of *Heterakis papillosa*, was removed from each of the parasitized birds.

A total of 136 birds were treated with different vermifuges. The number of worms each voided with the droppings during 24 to 48 hours following the administration of the vermifuge was determined, the bird then autopsied and the number of parasites and the species found in the intestine recorded.

The following vermifuges were tested:

1. Nicotin sulfate, in the form of Black Leaf 40, was administered in amounts ranging from 2.5 minims, which equaled one grain of nicotin sulfate, up to 1 cc (15 minims) of Black Leaf 40. In 30 birds, in which the maximum amount of Black Leaf 40 was 0.3 cc (plus 15 grains of kamala in each case) this combination proved to be 100 per cent efficient in removing the parasites from all parasitized birds, with the exception of cecal worms. In amounts above 0.3 cc, Black Leaf 40 did not prove so efficient and the birds receiving this drug in amounts of 0.5 to 1 cc very quickly showed the effects. In some cases the bird was prostrated within a few moments and in a few instances the birds died after receiving this amount of Black Leaf 40. In these cases the bird was very badly depressed when treated and probably would have died in a day or two.

2. Iodine vermicide, which is prepared according to the formula of Dr. W. L. Chandler, of Michigan State College, was very highly recommended in a paper presented at the December, 1931, meeting of the United States Live Stock Sanitary Association. This vermifuge was praised so highly that it was decided to test

TABLE I—*Summary of postmortem examinations.*

GROUP	SUB-GROUP	DATE TREATED	VERMIFUGE ADMINISTERED	BIRDS TREATED	BIRDS PARASITIZED	EFFICIENCY %
1	a	12–15–31	0.15 cc Black Leaf 40[1] (1 gr. nicotin sulfate) +15 gr. kamala	6	6	100
	b	12–15–31	0.3 cc Black Leaf 40 +15 gr. kamala	6	4	100
2	a	12–22–31	0.1 cc Black Leaf 40 +15 gr. kamala	6	3	100
	b	12–22–31	0.2 cc Black Leaf 40 +15 gr. kamala	6	4	100
3	a	12–29–31	0.3 cc Black Leaf 40 +15 gr. kamala	6	2	100
	b	12–29–31	0.4 cc Black Leaf 40 +15 gr. kamala	6	3	55+
4	a	1– 6–32	0.5 cc Black Leaf 40 +15 gr. kamala	6	2[2]	50
	b	1– 6–32	1.0 cc Black Leaf 40 +15 gr. kamala	6[3]	4	70
5		1–12–32	0.5 cc Black Leaf 40 +15 gr. kamala	10[4]	6[2]	?
6		2–13–32	0.5 cc Black Leaf 40 +15 gr. kamala	4	4	55
7		2–10–32	0.5 cc Black Leaf 40	8[5]	8	88
8		2–15–32	0.2 cc Black Leaf 40 +15 gr. kamala +0.4 gr. calomel	12[6]	4[2]	?
9		4–27–32	1 oz. iodine vermicide	12	11	60
10	a	4–30–32	1 oz. iodine vermicide	6	6	87.5
	b	4–30–32	1 Pulvule 142 (Lilly)	6	5[7]	97
11		5– 3–32	1 oz. iodine vermicide	6	5	75
12	a	5– 7–32	1 oz. iodine vermicide	6	5	75
	b[8]	5– 7–32	1 Pulvule 142 (Lilly) +15 gr. kamala	6	5	55
13	a	5–10–32	1 oz. iodine vermicide	6	4	91.5
	b	5–10–32	1 Pulvule 142 (Lilly) +15 gr. kamala	6	4	57.5
			Totals	136	95	

[1]2.5 minims (0.15 cc) of Black Leaf 40 contain 1 grain of nicotin sulfate.

[2]Parasites removed from one bird only.

[3]One bird died within ten hours after being treated.

[4]Three of this group of birds died within three hours following treatment.

[5]One of these birds died within a few hours following treatment. Was prostrated in less than 20 minutes.

[6]Two of these birds died within a few hours following treatment. All were heavily infested with lice and a number showed lesions of roup.

[7]Against *Ascaris lineata*.

[8]With this group of birds this vermifuge proved of little value.

it out in comparison with the one we found to be most efficient last year, namely, Pulvules No. 142 (Lilly), plus 15 grains of kamala. It was used on a total of 36 birds, with an efficiency of something more than 75 per cent.

3. Pulvules No. 142 (Lilly) (nicotin sulfate plus Lloyd's reagent) was used on a total of 18 birds and with fully as good results.

The cost of the iodine vermicide is only slightly less than four cents per bird, while the Pulvules, even when given in combination with 15 grains of kamala, would cost but 2¼ cents per bird.

Our conclusions are:

1. That Black Leaf 40, in doses of 0.1 to 0.3 cc, plus 15 grains of kamala, can be used safely and effectively for the removal of intestinal parasites from poultry at a very reasonable cost (two cents per bird or less).

2. That iodine vermicide and Pulvules No. 142 (Lilly), plus 15 grains of kamala, are of about equal efficiency but that the latter is less expensive.

3. That in heavily infested flocks, when there are many badly depressed, unthrifty birds, either iodine vermicide or Pulvules No. 142 (Lilly) could be used with greater safety.

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Canada Invites Visitors

Members of the A. V. M. A. attending the convention in Chicago, August 14-18, who may wish to include a trip to Canada in their itineraries, may obtain road maps, booklets and other pertinent information from the National Development Bureau of the Department of the Interior, Ottawa, Canada. The Bureau has available considerable unpublished data for those who make known their specific requirements concerning the districts in which they may be interested.

SPLENIC LESIONS IN HOG CHOLERA*

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INTRODUCTION

Recent investigations in the histopathology of hog cholera have aided immensely in explaining the development of the lesions observed. Fundamental work has been done on this phase of the disease by Roehrer[1] and Seifried and Cain.[2,3]

More attention than heretofore has been given to the lesions of the spleen by the investigators mentioned above. Characteristic macroscopic lesions, which have been described as hemorrhagic infarcts, frequently were found in this organ, particularly in virus-inoculated pigs. A detailed study of these lesions and their early development was deemed advisable.

MATERIAL AND METHODS

Spleens from 120 pigs, used for virus tests, were included in this study. The average weight was 60 pounds. Sixty pigs received a strain of virus designated as laboratory virus. This strain has been in use at this laboratory for approximately 25 years and its potency has been maintained from time to time by passage through very young pigs. The 59 other pigs received virus prepared by commercial establishments. Altogether 30 serials of virus, produced by 18 establishments, were given.

Each pig received 2 cc of virus in the axillary space. Temperatures and observations of the condition of the animals were recorded daily. Autopsy was performed at intervals between five and ten days following inoculation when the spleens and other desired tissues were obtained for study.

Spleens from 22 pigs, which were treated with cholera serum and a *Salmonella suipestifer* bacterin and later given an oral exposure of *S. suipestifer* organisms, were obtained. The animals were killed three weeks following the culture exposure. There were five anemic young pigs included in this group.

Examination was made of five spleens from pigs held for 14 days in a pen in which cholera pigs had been kept. Histologic studies were conducted in 19 field cases in which hog cholera was suspected, either from the history or the postmortem examination.

The spleens of seven pigs obtained from a hog cholera-free

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area in southern Indiana were designated as the "normal group." These spleens were from three lots of 75 pigs each. One lot was represented by three pigs and the two other lots by two pigs each.

Cultural examinations on beef-infusion agar were made on nearly all spleens and in some cases also the liver, heart and mesenteric lymph-glands. Sections were prepared by formol fixation and stained by the hematoxylin-eosin method, using Weigert's iron-hematoxylin on some sections and Harris' hematoxylin on others. Acid-fast stains were made on a number of splenic smears and also on sections showing typical lesions.

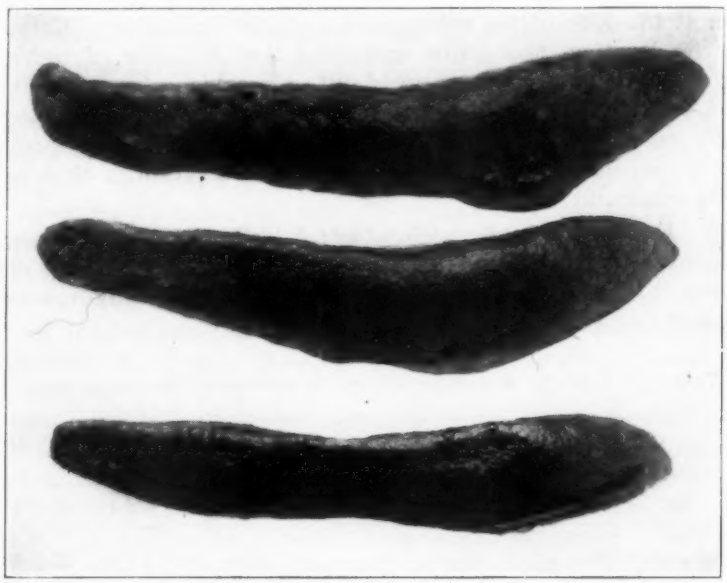

FIG. 1. Spleens showing formation of hemorrhagic areas not involving the margins of the organ.

DESCRIPTION OF LESIONS

For the purpose of describing the types of lesions observed histologically in the cases upon which this report is based, the following code is used:

A. Perifollicular hemorrhage: A zone of red blood cells located perivascularly in respect to the follicle artery.

B. Perifollicular necrosis: Necrobiotic areas occurring near the follicle artery. While these areas, when present, usually con-

tain some red blood cells, there is a more definite peripheral hemorrhagic zone.

C. Extensive necrosis of the parenchyma: Where the pulp and follicles are converted into a necrotic mass which contains cell débris, red blood cells and leucocytes. These areas apparently are the result of neighboring follicles becoming confluent. A definite peripheral hemorrhagic zone usually is obliterated, due to the extensive extravasation of blood.

D. Extreme hyperemia: The entire splenic pulp and sinuses are infiltrated with red blood cells, the follicles usually retaining their outlines.

E. Necrotic tip: An area of necrosis which, due to its location at the edge of the spleen, has a triangular outline. Except for its location, this lesion resembles the "C" type of lesion; the hemorrhage usually is spread diffusely, which gives the solid, dark-red appearance to these lesions when viewed macroscopically on cross-section. Necrosis of the capsule is infrequent, but a vacuolation suggesting hydropic degeneration is observed quite commonly.

F. Endothelioid cell proliferation: A striking increase of large mononuclear cells found immediately surrounding the follicular artery. Multinucleated forms and various mitotic figures are present quite frequently.

TABLE I—*Types of histologic lesions in spleens of various groups.*

GROUP	PIGS	NUMBER SHOWING TYPE LESIONS					
		A	B	C	D	E	F
Laboratory virus	61	59	39	8	4	22	58
Commercial virus	59	58	23	5	4	21	57
Experimental *S. suipestifer*	17	1	1	0	1	1	6
Experimental exposure	5	5	1	0	1	1	5
Field cases	19	14	1	2	6	3	14
Normal cases	7	0	0	0	1	0	3

RESULTS

Table I gives an analysis of the histological lesions observed in the various groups, and table II summarizes the pathological and bacteriological findings.

In the virus groups, numbering 120 pigs, there was found a temperature range between 103.0 and 108.0° F. High tempera-

tures were found in pigs showing slight lesions, and again, well-marked lesions were observed in pigs with low temperatures.

Vascular changes, indicated by hemorrhages of various degrees, were observed in all the spleens of virus-inoculated pigs. Necrotic lesions were more frequent in those receiving the laboratory strain of virus. A total of 31 spleens from both groups showed no macroscopic lesions, but microscopically they all showed vascular changes and necrotic lesions were present in six cases. No microörganisms were isolated from 111 spleens cultured. There were no acid-fast organisms found in the smears of 40 spleens equally divided between the two virus groups. Acid-fast stains of sections, showing typical lesions from each group, also were negative.

S. suipestifer was recovered from three spleens of the group which was given this organism by the mouth following a bacterin treatment. One spleen was from an anemic pig, which showed the characteristic picture of anemia microscopically. The two other spleens showed a few endothelioid cells in the follicles. One spleen, from which no organism was isolated, showed the vascular and necrotic lesions observed in the virus pigs. With the exception of the spleens from the anemic pigs, all of which showed various degrees of hematopoiesis, there were no lesions in the remaining spleens.

All the spleens from the pigs given a pen exposure showed characteristic lesions observed in virus pigs, although not so pronounced.

Macroscopic lesions were observed in one field case. Vascular changes were observed microscopically in 17 of the 19 cases, and five showed lesions of necrosis. Two cases yielded *S. suipestifer*.

No macroscopic lesions were observed in the spleens of pigs from the cholera-free locality. One spleen showed a hyperemia upon microscopic examination. All cultures from this group were negative.

Discussion

It appears that lesions in cholera spleens, characterized by a collection of endothelioid cells adjacent to the follicle artery, with an immediate outer zone of hemorrhage, indicate the development of the more extensive changes leading to the formation of necrotic areas. Figure 2 illustrates a typical early stage in the development of a necrotic area in the edge of the spleen.

The endothelioid cell proliferation (figs. 3 and 4) is a striking feature in the virus-inoculated pigs and also the field cases of hog cholera. Slight cellular reactions are observed in some of

TABLE II—*Summary of histopathological and bacteriological examination of spleens.*

GROUP	PATHOLOGICAL DATA						BACTERIOLOGICAL DATA		
	PIGS	VASCULAR		NECROTIC		NECROTIC AND VASCULAR	PIGS EXAMINED	SALMONELLA SUIPESTIFER	OTHER ORGANISMS
		No.	%	No.	%				
Laboratory virus	61	61	100.0	47	77.0	47	61	0	0
Commerical virus	59	59	100.0	34	57.6	34	50	0	0
Experimental *S. suipestifer*	17	1	5.8	1	5.8	1	17	3	0
Experimental exposure	5	5	100.0	1	20.0	1	0	0	0
Field cases	19	17	89.4	5	26.3	5	15	2	0*
Normal	7	1	14.2	0	0	0	7	0	0

**E. coli* types isolated in a few instances were considered as contaminants.

the spleens from the pigs on the *S. suipestifer* experiment and also in three spleens from pigs of the "normal" group. The pigs used for virus tests at this laboratory are between three and four months of age. Lymphoid hyperplasia, observed in the splenic follicles of young animals, must be borne in mind when weighing the significance of cellular reactions. The marked proliferation in cholera spleens, however, is not observed in normal cases.

Megakaryocytes, also seen in the spleens of young subjects, are observed in some spleens of the anemic pigs which are between eight and ten weeks of age. These cells are not observed in the older pigs. There should be no confusion between the megakaryocytes and the endothelioid cells described in this paper,

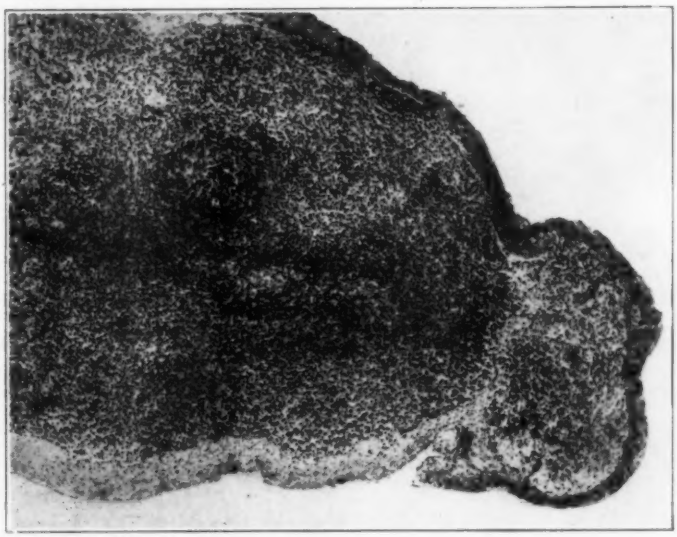

FIG. 2. Early lesion of endothelioid cells and hemorrhage around follicle arteries in marginal area of organ. No macroscopic lesions. Acid-fast stain negative.

since the former have an entirely different morphology and the distribution is not confined to the follicle arteries.

Necrobiotic foci (fig. 5), sometimes quite small, become confluent with neighboring foci resulting in a large necrotic area. The small perifollicular type of necrotic lesion, designated as the "B" lesion, is much more frequent in virus spleens than the "C" type and also occurs more frequently than the "E" type, the necrotic tip.

Pronounced alterations in the walls of arteries sometimes are seen (fig. 6). The walls are permeable to the red blood cells,

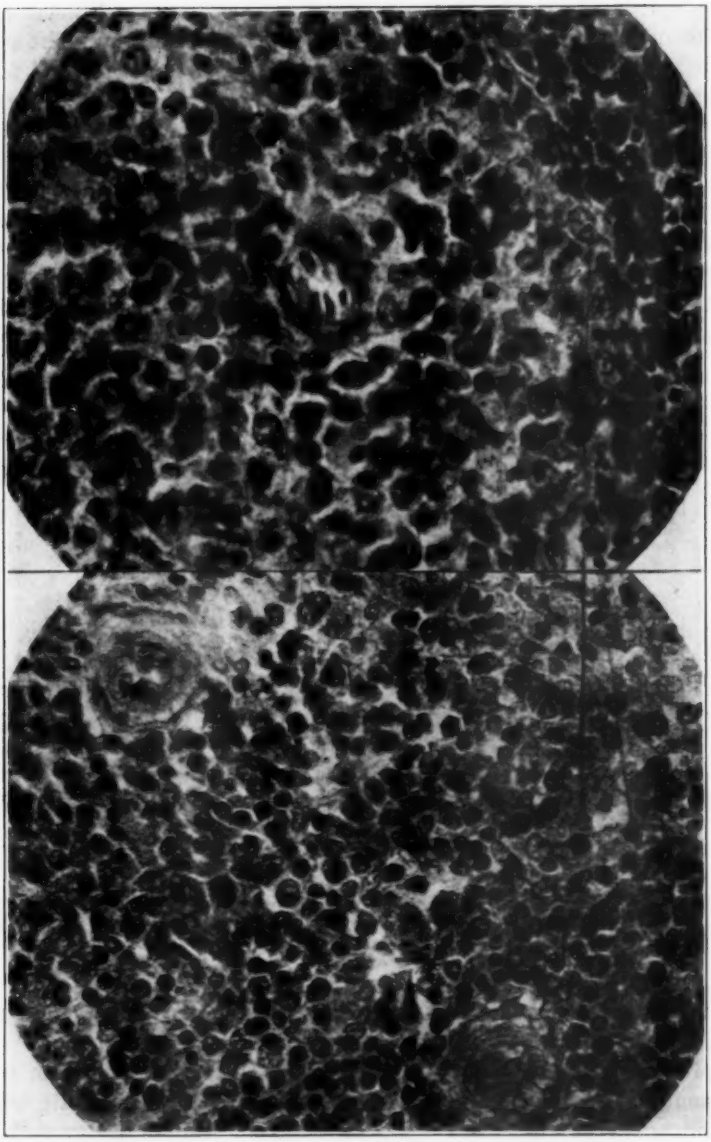

FIG. 3 (above). Large endothelioid cells with numerous mitotic figures surrounding follicular artery. No macroscopic lesions. Acid-fast stain negative.

FIG. 4 (below). Endothelioid cells in follicle showing mononuclear and multinucleated forms. No macroscopic lesions. Acid-fast stain negative.

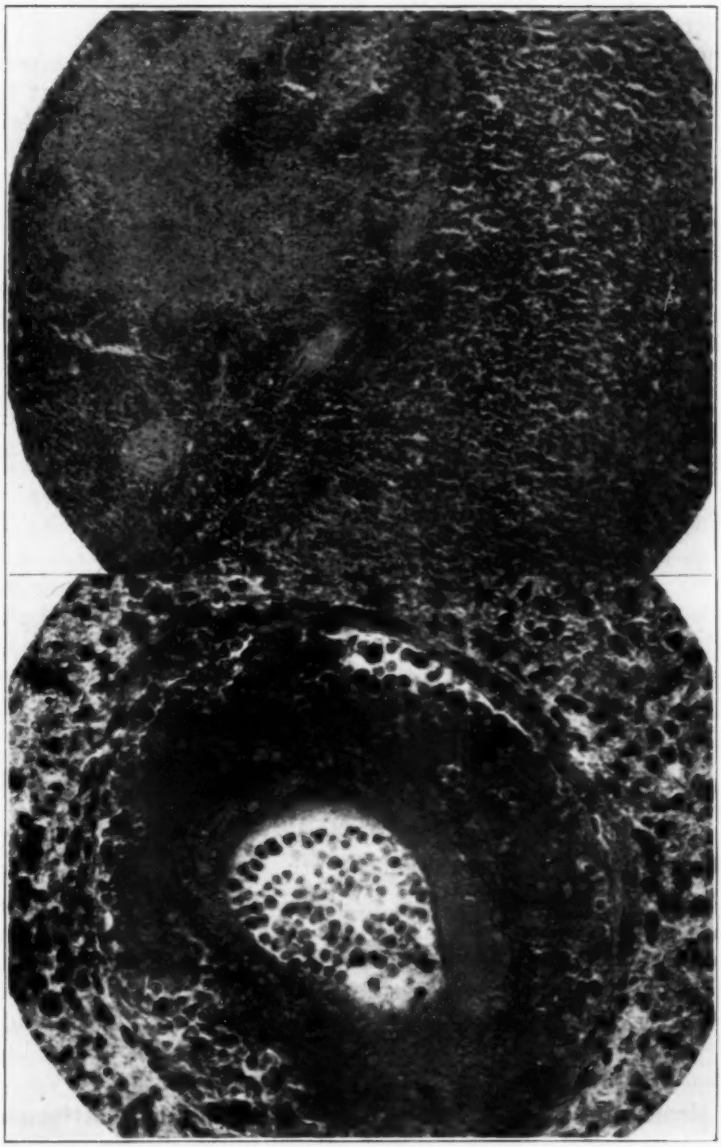

FIG. 5 (above). Necrobiotic focus adiacent to follicle artery. Macroscopic lesions evident. Acid-fast stain negative.

FIG. 6 (below). Larger artery showing hyalinized intima; wall shows marked alterations. Lesions evident macroscopically. Acid-fast stain negative.

which probably accounts for the extensive hemorrhage in the necrotic areas.

Two field cases, in which *S. suipestifer* infection is present, show rather pronounced vascular and necrotic changes, but just as severe alterations are found in spleens in which there is no secondary infection.

SUMMARY

Hemorrhagic and necrotic lesions are found in hog-cholera spleens independent of secondary infection.

Early lesions in the spleen are characterized by an endothelioid cell proliferation adjacent to or surrounding the follicular arteries, with an outer zone of hemorrhage.

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Reduced Railway Fares to Chicago

The reduced railroad rates that are now in effect all over the country should offer a special inducement to members of the American Veterinary Medical Association who are planning to attend the convention in Chicago, August 14-18. Members may enjoy a delightful trip from the East, West, North or South, attend the meeting and take in the Century of Progress at a fair and reasonable price. The railroads promise that.

Round-trip tickets may be bought at prices ranging from a fare and a half to a fare and a tenth, depending upon the time the individual plans to take for his trip.

It would be well, therefore, for members to see their local railroad agents, study the different rates offered, and make plans accordingly. Some railroads are offering a round-trip season ticket, good until November 15, for a fare and a half, and a 30-day round-trip ticket for a fare and a third. A 16-day-limit round-trip ticket may be had for a fare and a tenth.

Members who may say, "I'd like to attend the meeting, but the trip costs too much," would do well to talk things over with their local ticket agents.

Come to Chicago—August 14-15-16-17-18

STUDIES OF THE SKIN AS A PORTAL OF ENTRY FOR BRUCELLA ABORTUS IN PREGNANT CATTLE*

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The portals of entry for *Brucella abortus* into cattle to cause Bang's disease have been matters that have frequently engaged the attention of investigators since the discovery by Bang,[1] in 1897, of the causative microörganism. The ability of *Br. abortus* of normal virulence to set up disease when introduced in sufficient amounts into the digestive or genital tracts of pregnant animals has been demonstrated repeatedly. More recent investigations, which we have conducted, also have revealed the ability of *Br. abortus*, when deposited in the conjunctival sac, even in comparatively small amounts, likewise to induce the disease. The idea that the skin also might constitute a portal of entry for the infection had received but scant attention until Hardy and his associates[2] conducted transmission experiments with guinea pigs which resulted in such a manner as to cause them to conclude that even the intact skin was more vulnerable as a portal of entry for the infection in those animals than the digestive tract. The results obtained by Hardy and his co-workers prompted efforts on our part to determine the effects of *Br. abortus* skin exposure upon pregnant, susceptible cattle.

Studies of this subject were commenced in 1929. The results that were derived from the subjection of four pregnant heifers to skin exposure were described by one of us (W. E. C.)[3] at the Eleventh International Veterinary Congress, at London, in 1930. Further results which the investigation yielded were presented at the 1930[4] and 1931[5] meetings of the United States Live Stock Sanitary Association.

Oluf Bang,[6] in a recent publication, describes investigations which deal with the transmission of the disease through the skin. He reports success in transmitting the malady to seven cattle through the intact skin and to two through the scarified skin of the teats, by using emulsions of *Br. abortus*-infected placenta and culture suspensions. The infectious material was applied in seven cases to shaved areas of the fetlock.

In our investigations of this subject, use has thus far been made of 18 pregnant heifers and two pregnant cows. Breeding

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records of the animals were not available. They were in the early stage of pregnancy when purchased and delivered at the Experiment Station, Bethesda, Md. Pregnancy examinations, however, were made shortly before they were placed in the experiment.

A small area of the skin of each of three heifers and one cow, to which *Br. abortus* was applied, was shaved and slightly scarified at the time of exposure. The infection was applied to the intact skin of 15 heifers and one cow. The infection usually was applied to a single area of the skin, from four to six inches in diameter, in the gluteal region or slightly posterior to the shoulder. The infection was applied to the teats of one cow used in the experiment.

In all cases the bovine type of *Br. abortus* was used in making the exposures. The suspensions applied to the skin always were prepared from *Br. abortus* strains recently isolated from aborting cows and could reasonably be expected to be of normal virulence. The cultures from which the suspensions were prepared were never more than 72-hour transfers. All of the suspensions, at the time of use, were proved to be virulent for guinea pigs. The *Br. abortus* suspensions ranged in density between 10 and 60 times that of tube 1 of the McFarland nephelometer. Usually about 10 cc of a suspension was used in giving an animal a single exposure. A period of about five minutes generally was used in applying and rubbing the suspensions on the skin with a cotton swab. In most of the cases in which infection was attempted through the intact skin, the hair was clipped with scissors from the area to be exposed, but in a few instances the infection was applied without the removal of any of the hair. When the hair was clipped, great care was taken to avoid injury to the skin. Pledgets of cotton moistened with the suspensions were applied to the skin and covered with rubber-cloth patches in a few instances and allowed to remain as long as the patches adhered, it being thought that prolonged contact might increase the chances of the infectious agent passing through the skin.

In order to exclude as far as possible other portals of entry than the one under consideration, the following precautions were taken. The stanchions in which most of the animals were confined continuously were so constructed as to prevent any possibility of contact between their mouths and portions of their bodies posterior to their necks. The manner in which they were constructed also largely overcame the possibility of bedding or other materials passing from the floors of the stalls to the man-

gers, as well as placing beyond the reach of the animals any feed that might come in contact with their feet or portions of their bodies other than their heads and necks. The areas of the skin to which *Br. abortus* was applied were, in all but one case (in which exposure was made on the teats), covered with patches of heavy cotton cloth held in position with adhesive tape or rubber-cloth patches, to the edges of which tar was applied to cause their adherence to the skin. The patches were about ten inches in diameter. It was frequently possible to keep the exposed areas covered by the patches for two weeks or longer. The precautions that were taken to prevent the disease being transmitted through other channels than the one being studied (the skin) gave every indication of being effective.

A single application of a *Br. abortus* saline suspension to the slightly abraded skin resulted in the development of agglutination reactions in all four animals which were subjected to this type of exposure. One of these four animals had failed previously to react to the agglutination test, following repeated applications of freshly drawn milk which contained *Br. abortus* to slightly abraded surfaces of the teats. When *Br. abortus* suspensions were applied to the intact skin, the results were less uniform than when such suspensions were applied to the abraded skin areas. If, after a period of from one to three months, the absence of invasion of the infection was indicated by negative agglutination results or reactions of low intensity with the blood of the exposed animal, a second skin exposure was made, usually with a different strain or strains of *Br. abortus*. One heifer was exposed even a third time. More detailed information as to the procedure, which was followed with each animal, and the results obtained follows:

ANIMALS EXPOSED THROUGH THE ABRADED SKIN

Heifer 1583: Estimated to be approximately 5½ months pregnant, November 15, 1929. Agglutination test, November 24, was negative. Applied to a slightly abraded skin area in the gluteal region, November 25, about 5 cc of *Br. abortus* suspension, 30 times density of tube 1 of the McFarland nephelometer, which was prepared from *Br. abortus* strains 1456 (fifth transfer), 813 (first transfer), and 1497 (eighth transfer). Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: December 19, 1:100; January 8, 1930, 1:100; January 22, 1:200; and February 5, 1:200. Gave birth to a weak calf, February 18. Placenta showed lesions suggestive of *Br. abortus* infection. Guinea pigs inoculated with an emulsion of placenta acquired abortion disease, as did also guinea pigs which were inoculated with colostrum.

Heifer 1587: Estimated to be approximately 4 months pregnant, November 15, 1929. Agglutination test, November 24, was negative.

Subjected to *Br. abortus*, abraded-skin exposure, November 25, using a portion of the same lot of *Br. abortus* suspension as was used for the exposure of heifer 1583. Agglutination tests: December 19, 1:200; January 8, 1930, 1:1,000; January 22, 1:1,000; February 5, 1:2,000; and February 19, 1:4,000. Gave birth to a weak calf, February 24, which soon died. Placenta showed characteristic lesions of *Br. abortus* infection. *Br. abortus* isolated from placenta and colostrum through guinea-pig inoculations.

Heifer 1722: Estimated to be about 5 months pregnant, May 20, 1932. Agglutination test, May 10, was negative. Applied a suspension of *Br. abortus*, with a density 25 times tube 1 of the McFarland nephelometer, to a slightly scarified area of the skin in the gluteal region, May 23. The suspension was prepared from *Br. abortus* strains 674 (third transfer) and 695 (second transfer). Exposed area was covered with a rubber cloth patch which was held in position by tar applied to its edges. Agglutination tests: May 24, negative: June 1, negative; June 11, 1:200; June 27, 1:500; and July 23, 1:1,000. Gave birth to what appeared to be about an 8-month calf, August 8. Placenta passed promptly and normal in appearance. *Br. abortus* isolated from colostrum through guinea-pig inoculations but guinea pigs inoculated with placental emulsion remained healthy.

Cow 1670: Estimated to be about 5 months pregnant, February 20, 1932. Agglutination test, February 29, was negative. Small areas of the skin on all four teats were scarified slightly, February 29, and freshly drawn milk of two cows that were proved to be eliminating *Br. abortus* in their milk immediately applied. Other applications of freshly drawn milk from the same two cows were made to the slightly abraded skin areas of the teats as follows: Twice, March 4; once, March 7; once, March 8; once, March 10; twice, March 11; once, March 12; and once, March 15. Agglutination test, March 25, was negative. Because of failure to acquire a reaction to the agglutination test, a suspension 25 times the density of tube 1 of the McFarland nephelometer, prepared from *Br. abortus* strain 19 (second transfer), was applied to a small scarified area on one teat, April 1. Agglutination tests, April 5, April 14, May 10, and May 24, were negative. Agglutination tests: June 1, 1:25; and June 11, 1:200. Cow gave birth to a weak calf, June 23, which soon died. Placenta retained. *Br. abortus* isolated from uterine exudate and from colostrum through guinea-pig inoculations. Agglutination test, June 23, 1:1,000.

Table I shows in condensed form the results following abraded-skin exposure to *Br. abortus*.

TABLE I—*Summary of results obtained from Brucella abortus, abraded-skin exposure.*

ANIMAL	NUMBER OF APPLICATIONS OF BR. ABORTUS	OUTCOME OF PREGNANCY	RESULTS OF UTERINE EXAMINATIONS FOR BR. ABORTUS	RESULTS OF COLOSTRUM EXAMINATIONS FOR BR. ABORTUS
Heifer 1583	1	Weak calf	+	+
Heifer 1587	1	Weak calf	+	+
Heifer 1722	1	Weak calf	−	+
Cow 1670	10, *Br. abortus*—infected milk; 1, *Br. abortus*—saline suspension	Weak calf	+	+

ANIMALS SUBJECTED TO INTACT SKIN EXPOSURE

Heifer 1594: Estimated to be about 4 months pregnant, February 6, 1930. Agglutination test, February 1, was negative. On February 8, applied about 10 cc of a *Br. abortus* suspension, 60 times density of tube 1 of the McFarland nephelometer, to an unclipped area of skin in the gluteal region. The suspension was prepared from *Br. abortus* strain 1429 (first transfer). Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: February 24, 1:100; March 5, 1:1,000; March 19, 1:1,000; and April 12, 1:1,000. Heifer aborted, April 28. Placenta showed typical lesions of *Br. abortus* infection. *Br. abortus* isolated from fetus by cultural methods and from the colostrum through guinea-pig inoculations.

Heifer 1602: Estimated to be about 5 months pregnant, February 6, 1930. Agglutination test, February 6, was negative. This heifer was subjected to intact-skin exposure, on the same date as heifer 1594, use being made of a portion of the same lot of *Br. abortus* suspension. Previous to the application of the suspension in this case, however, the hair was partially removed from the area with scissors. The exposed area then was covered with a heavy, cotton-cloth patch. Agglutination tests: February 24, 1:100; March 5, 1:200; March 19, 1:100; and April 12, 1:100. Heifer gave birth to a weak calf, May 21. Guinea pigs inoculated with uterine material acquired abortion disease but guinea pigs inoculated with colostrum remained healthy.

Heifer 1555: Estimated to be about 5½ months pregnant, April 6, 1930. Agglutination test, April 7, was negative. Was subjected to intact-skin exposure, April 8, use being made of a *Br. abortus* suspension having a density 10 times that of tube 1 of the McFarland nephelometer, prepared from *Br. abortus* strain 1446 (fourth transfer). The suspension was applied to an unclipped area of skin in the gluteal region. Exposed area was covered with a heavy, cotton cloth patch. Agglutination tests, April 15, April 22, and May 7, were negative. Agglutination tests: May 17, 1:25; June 2, 1:1,000; June 17, 1:1,000; June 28, 1:1,000; and July 16, 1:1,000. Animal gave birth to a vigorous calf, August 6. Placenta passed promptly. Guinea pigs inoculated with uterine material failed to acquire abortion disease. *Br. abortus* isolated from colostrum through guinea-pig inoculations.

Heifer 1460: Estimated to be about 5½ months pregnant, April 4, 1930. Agglutination test, April 7, was negative. This heifer was subjected to intact-skin exposure, April 8. The hair from an area in the gluteal region was clipped with scissors. To this area was applied a portion of the same *Br. abortus* suspension that was used to expose heifer 1555. Exposed area then was covered with a heavy, cotton-cloth patch. Agglutination tests, April 15, April 22, and May 7, were negative. Inasmuch as heifer 1460 failed to acquire a reaction to the agglutination test, it was decided to expose her a second time. On May 10, approximately 10 cc of a *Br. abortus* suspension, 20 times the density of tube 1 of the McFarland nephelometer, was applied to an area of unclipped skin in the gluteal region. The suspension was prepared from *Br. abortus* strain 1594 (first transfer). Agglutination tests, May 17, June 2, June 17, June 28, July 16, and August 19, were negative. Animal gave birth to a vigorous calf, July 30. Guinea pigs inoculated with uterine material and with colostrum failed to acquire abortion disease.

Heifer 1457: Estimated to be about 3 months pregnant, April 4, 1930. Agglutination test, April 7, was negative. This animal was subjected to intact-skin exposure on the same date and in like manner as heifer 1555, use being made of a portion of the same *Br. abortus* suspension. Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: April 15, negative; April 22, negative; May 7,

1:25; and May 17, June 2, June 17, June 28, July 16, August 19, and October 2, all negative. This animal gave birth to a vigorous calf, October 14. Guinea pigs inoculated with uterine material as well as those inoculated with colostrum failed to acquire abortion disease.

Heifer 1326: Estimated to be about 4 months pregnant, April 4, 1930. Agglutination test, April 7, was negative. This heifer was subjected to intact-skin exposure on the same date and in like manner as heifer 1555, use being made of a portion of the same *Br. abortus* suspension. Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests, April 15, April 22, and May 7, were negative. Since this animal remained negative to the agglutination test, she was subjected to a second skin exposure, May 10. Approximately 10 cc of a *Br. abortus* suspension, 20 times the density of tube 1 of the McFarland nephelometer, was applied to an unclipped area of skin in the gluteal region. The suspension was prepared from *Br. abortus* strain 1599 (first transfer). Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: May 17, 1:25; June 2, negative; June 17, 1:200; June 28, 1:200; July 16, 1:500; and October 2, 1:8,000. This animal gave birth to a weak calf, August 16. The placenta was retained. Guinea pigs inoculated with uterine material, as well as those inoculated with colostrum, acquired abortion disease.

Heifer 1610: Estimated to be about 4 months pregnant, April 4, 1930. Agglutination test, April 7, was negative. This animal was subjected to intact-skin exposure on the same date and in the same manner as heifer 1555, use being made of a portion of the same *Br. abortus* suspension. Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests, April 15, April 22, and May 7, were negative. Animal was subjected to a second skin exposure, May 10. Approximately 10 cc of a suspension, 20 times the density of tube 1 of the McFarland nephelometer and prepared from *Br. abortus* strain 1594 (first transfer), was applied to a clipped area in the gluteal region. Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: May 17, 1:25; June 2, June 17, June 28, July 15, August 19 and October 2, negative. Animal produced a vigorous calf, September 2. Guinea pigs inoculated with uterine material and with colostrum failed to acquire abortion disease.

Heifer 1582: Estimated to be about 2 months pregnant, January 10, 1931. Agglutination test, January 10, was negative. Was subjected to intact-skin exposure, January 14. Approximately 8 cc of a *Br. abortus* suspension, 50 times the density of tube 1 of the McFarland nephelometer, was applied to an area of the skin posterior to the left shoulder from which the hair had been clipped with scissors. The suspension was prepared from *Br. abortus* strain 1432 (first transfer). A patch of heavy, cotton cloth was used to cover the exposed area. Agglutination tests: February 3, 1:100; February 19 and March 2, negative. Animal was subjected to a second skin exposure, April 1. The suspension employed had a density of about 20 times tube 1 of the McFarland nephelometer and was prepared from *Br. abortus* strain 1542 (first transfer). Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: April 29, 1:25; May 17, June 2, June 17, and June 28, negative. Heifer was subjected to a third skin exposure, July 9. At this time the suspension, having a density 30 times tube 1 of the McFarland nephelometer, was prepared from the fifth transfers of *Br. abortus* strains 1542 and 1595. It was applied to a clipped area of skin posterior to the left shoulder. Agglutination tests: July 23, August 12, August 22, September 9, October 15, and October 23, negative. Animal gave birth to a vigorous calf, August 24. Guinea pigs inoculated with uterine material and with colostrum failed to acquire abortion disease.

Heifer 1585: Estimated to be about 4 months pregnant, July 1, 1931. Agglutination test, July 7, was negative. Applied to a clipped area posterior to right shoulder, July 9, approximately 10 cc of a *Br. abortus* suspension 30 times as dense as tube 1 of the McFarland nephelometer. The suspension was prepared from *Br. abortus* strains 1542 and 1595 (fifth transfers). Exposed area was covered with a heavy, cotton-cloth patch. Agglutination tests: July 23, negative; August 12, 1:25; August 22, 1:25; September 9, 1:200; October 15, 1:1,000; and October 23, 1:1,000. Animal aborted, September 11. Placenta retained. *Br. abortus* isolated from fetus by cultural methods. Guinea pigs inoculated with colostrum acquired abortion disease.

Cow 1672: Estimated to be about 3 months pregnant, October 25, 1931. Agglutination test, October 27, was negative. Subjected to intact-skin exposure, October 29. Approximately 10 cc of a *Br. abortus* suspension, 20 times the density of tube 1 of the McFarland nephelometer, was applied to a clipped area of the skin in the gluteal region. The suspension was prepared from *Br. abortus* strain A 52 (second transfer). Exposed area was covered with a rubber-cloth patch. Agglutination tests: November 5, negative; November 28, 1:50; January 6, and January 12, 1932, negative. Animal was subjected to a second skin exposure, January 12. Approximately 10 cc of a *Br. abortus* suspension, 15 times the density of tube 1 of the McFarland nephelometer, was applied to an area of the clipped skin in the gluteal region. The suspension was prepared from *Br. abortus* strain 1001 (first transfer). In making this exposure, a pledget of absorbent cotton was moistened with the suspension, applied to the clipped area, and held in position by a rubber-cloth patch. Agglutination tests: January 28, 1:200; March 10, 1:200; March 25, 1:500; April 5, 1:200; and April 14, 1:500. Animal gave birth to a vigorous calf, May 1. Guinea pigs inoculated with uterine material acquired abortion disease as did those also which were inoculated with colostrum.

Heifer 1701: Estimated to be about 4 months pregnant, October 25, 1931. Agglutination test, October 27, was negative. Was subjected to intact-skin exposure, October 29, when approximately 10 cc of a *Br. abortus* suspension, 20 times the density of tube 1 of the McFarland nephelometer, was applied to the skin in the gluteal region from which the hair had been clipped. The suspension was prepared from *Br. abortus* strain A-52 (second transfer). Exposed area was covered with a rubber-cloth patch. Agglutination tests: November 5, negative; November 28, 1:50; January 6, 1932, negative. Animal was subjected to a second skin exposure, January 13. Approximately 10 cc of a *Br. abortus* suspension was applied to the skin in the gluteal region from which the hair had not been clipped. The suspension was prepared from *Br. abortus* strain 1001 (first transfer), and had a density of 15 times that of tube 1 of the McFarland nephelometer. A pledget of absorbent cotton moistened with the suspension then was applied to the skin, and held in place by a rubber-cloth patch. Agglutination tests: January 28, 1:200; March 10, 1:1,000; and March 25, 1:1,000. Animal gave birth to a vigorous calf, March 20. *Br. abortus* isolated from placenta by cultural methods. Guinea pigs inoculated with uterine exudate acquired abortion disease as did those inoculated with colostrum.

Heifer 1483: Estimated to be about 5 months pregnant, December 16, 1931. Agglutination test December 20, was negative. Was subjected to intact skin exposure, December 21. Approximately 10 cc of a *Br. abortus* suspension was applied to an area of the skin in the gluteal region from which the hair had been clipped. The suspension was prepared from *Br. abortus* strain A-52 (sixth transfer), and had a density of 40 times that of tube 1 of the McFarland nehelometer. Ex-

posed area was covered with a rubber-cloth patch. Agglutination tests: January 6, 1932, 1:100; January 12, 1:50; January 28, 1:25; February 23, 1:200. Animal was subjected to a second skin exposure, February 23, before the results of the agglutination test with blood obtained on the same day were ascertained. A *Br. abortus* suspension, 10 times the density of tube 1 of the McFarland nephelometer and prepared from *Br. abortus* strain 1001 (fourth transfer), was applied to an area of the skin in the gluteal region from which the hair had been clipped. Agglutination test, March 10, 1:4,000. Animal gave birth to a weak calf, March 9, which failed to survive. Placenta retained. *Br. abortus* isolated from calf by cultural methods. Guinea pigs inoculated with uterine material acquired abortion disease but those inoculated with colostrum remained healthy.

Heifer 1518: Estimated to be about 7 months pregnant, January 10, 1932. Agglutination test, January 10, was negative. Was subjected to intact-skin exposure, January 12. Approximately 10 cc of a suspension prepared from *Br. abortus* strain 1001 (first transfer), and having a density of 15 times tube 1 of the McFarland nephelometer, was applied to a clipped area of the skin in the gluteal region. Exposed area, to which a pledget of absorbent cotton saturated with the suspension was applied, was then covered with a rubber-cloth patch. Agglutination tests, January 28, 1:50; February 23, negative. Animal was subjected to a second skin exposure, February 26, when a suspension prepared from *Br. abortus* strain 1001 (fifth transfer) was applied to an area of the skin anterior to the external angle of the ilium from which the hair had been clipped. The method was the same as that used in the first exposure. The density of the suspension was 10 times that of tube 1 of the McFarland nephelometer. Agglutination tests: March 10, 1:50; March 15, 1:50; and April 5, 1:100. Animal gave birth to a vigorous calf, March 14. *Br. abortus* isolated from both uterine material and colostrum through guinea pig inoculations.

Heifer 1731: Estimated to be about 4 months pregnant, February 20, 1932. Agglutination test, February 20, was negative. Was subjected to intact-skin exposure, February 23. Approximately 10 cc of a *Br. abortus* suspension prepared from *Br. abortus* strain 1001 (fourth transfer), and having a density of 10 times that of tube 1 of the McFarland nephelometer, was applied to a clipped area of the skin in the gluteal region. A pledget of absorbent cotton saturated with the suspension was also applied to the cliped area, and held in position with a rubber-cloth patch. Agglutination tests: March 10, 1:25; March 16, 1:25; March 25, negative. Animal was subjected to a second skin exposure, April 1, in the same region and in the same manner. The suspension used had a density of 25 times that of tube 1 of the McFarland nephelometer and was prepared from *Br. abortus* strain 1483 (second transfer). Agglutination tests: April 5, negative; April 14, negative; May 10, 1:200; May 24, 1:200; June 1, 1:200; June 11, 1:200; July 23, 1:1,000. Animal gave birth to a weak calf, June 25. *Br. abortus* isolated from uterine material and colostrum through guinea pig inoculations.

Heifer 1745: Estimated to be about 3 months pregnant, February 20, 1932. Agglutination test, February 20, was negative. Was subjected to intact-skin exposure, February 23, the same method and locations being used as in the case of heifer 1731. Use was made of approximately 10 cc of a suspension of *Br. abortus* prepared from strain 1001 (fourth transfer), and having a density of 10 times that of tube 1 of the McFarland nephelometer. Agglutination tests: March 10, 1:200; March 16, 1:200; March 25, 1:100; April 5, 1:50; April 14, 1:50; and May 10, 1:50. Animal was subjected to a second skin exposure, May

23. The suspension used was prepared from *Br. abortus* strains 674 (third transfer) and 695 (second transfer), and had a density of 25 times that of tube 1 of the McFarland nephelometer. The suspension was applied as in the previous exposure. Agglutination tests: May 24, 1:50; June 1, 1:50; June 11, 1:50; July 23, 1:50; and August 14, 1:200. Animal gave birth to a vigorous calf, August 19. Guinea pigs inoculated with uterine material as well as those inoculated with colostrum failed to acquire abortion disease.

Heifer 1725: Estimated to be about 6 months pregnant, March 25, 1932. Agglutination test, March 28, was negative. Was subjected to intact-skin exposure, April 1, in the same manner and in the same locations as in the case of heifer 1731. Approximately 10 cc of a *Br. abortus* suspension, 25 times the density of tube 1 of the McFarland nephelometer, and prepared from *Br. abortus* strain 19 (second transfer), was used. Agglutination tests: April 5, negative; April 14, 1:100; May 10 and May 20, negative. Animal was subjected to a second skin exposure, May 23, by the same method and in the same location. Approximately 10 cc of a *Br. abortus* suspension, prepared from *Br. abortus* strains 674 (third transfer) and 695 (second transfer), was applied. This suspension had a density of 25 times that of tube 1 of the McFarland nephelometer. Agglutination tests, June 1, June 11, June 23, and August 7, were negative. Animal gave birth to a vigorous calf, June 29. Guinea pigs inoculated with uterine material and those inoculated with colostrum failed to acquire abortion disease.

Table II gives the summary of the results derived from intact-skin exposure to *Br. abortus*.

DISCUSSION

The results of the experiment indicate that pregnant, susceptible cattle may be readily infected with Bang's disease by the

TABLE II—*Summary of results obtained from Brucella abortus intact-skin exposure.*

ANIMAL	NUMBER OF APPLICATIONS OF BR. ABORTUS	OUTCOME OF PREGNANCY	RESULTS OF UTERINE EXAMINATIONS FOR BR. ABORTUS	RESULTS OF COLOSTRUM EXAMINATIONS FOR BR. ABORTUS
Heifer 1594	1	Abortion	+	+
Heifer 1602	1	Weak calf	+	−
Heifer 1555	1	Vigorous calf	−	+
Heifer 1460	2	Vigorous calf	−	−
Heifer 1457	1	Vigorous calf	−	−
Heifer 1326	2	Weak calf	+	+
Heifer 1610	2	Vigorous calf	−	−
Heifer 1582	3	Vigorous calf	−	−
Heifer 1585	1	Abortion	+	+
Cow 1672	2	Vigorous calf	+	+
Heifer 1701	2	Vigorous calf	+	+
Heifer 1483	2	Weak calf	+	−
Heifer 1518	2	Vigorous calf	+	+
Heifer 1731	2	Weak calf	+	+
Heifer 1745	2	Vigorous calf	−	−
Heifer 1725	2	Vigorous calf	−	−

application of *Br. abortus* suspensions to a slightly abraded surface of the skin. When *Br. abortus* suspensions were applied to intact skin areas the disease was conferred less regularly. It was not always possible to transmit the disease through the intact skin even when animals were subjected to two exposures. It is not certain as to the bearing that the second skin exposures had upon the transmission of the disease because of the absence of definite information regarding the incubation period. Following the application to the intact skin of *Br. abortus* suspensions, low reactions to the agglutination test frequently were obtained in from two to three weeks, although the reactions in a number of cases later subsided or disappeared altogether.

SUMMARY

Four pregnant cattle were used to test the effects of abraded-skin exposure to *Brucella abortus* (bovine) and sixteen pregnant cattle were used to test the effects of intact-skin exposure.

Following the application of *Br. abortus* suspensions to a small area of the abraded skin of four cattle, Bang's disease was conferred in all cases.

Bang's disease was transmitted to ten of sixteen pregnant cattle following the application of *Br. abortus* suspensions to small areas of the intact skin.

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Finding a Chair at the World's Fair

Chairs are usually one of the scarcest commodities at expositions. But not at the Chicago Century of Progress Exposition. No less than 75,000 chairs will be distributed about the fair grounds, so that visitors may rest between spurts of sightseeing —and there will be no charge. Huge bright-colored umbrellas, each large enough to shelter forty persons, will cover benches placed conveniently about the grounds. And each of the major buildings will have a lounge room, with comfortable chairs, desks, writing paper and cold drinking water.

ANAPLASMOSIS

V. The Nature of Anaplasma*

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There are at the present time three prevalent opinions concerning the nature of *Anaplasma*, as follows: (*a*) That they are a phase in the life-history of some member of the Piroplasmidae. (*b*) That they are "Jolly bodies" or reaction products, produced by an invisible, unfiltrable virus and analogous to the Jolly bodies produced in other animals by splenectomy or the injection of such substances as pyrogallic acid, phenylhydrazine, etc. (*c*) That they are true endoglobular parasites, *sui generis*, and as commonly recognized and described under that name.

A large amount of literature[30, 31] discussing these points has accumulated since Theiler in 1910, described the disease anaplasmosis and advanced the opinion that the "marginal points," Anaplasma, were the causative agents. Without going into an exhaustive review of the literature, it may be stated that as far as we are concerned, we can safely dispose of the first opinion, *viz.*, that Anaplasma is a phase of the life history of another member of the Piroplasmidae. As far as we know today, only one species of Piroplasma occurs in cattle in the United States, and bovine piroplasmosis in the United States does not occur in the absence of its vector, *Boöphilus annulatus*. Our cases in Louisiana, diagnosed as anaplasmosis, have occurred in areas officially declared tick-free and released from quarantine. Anaplasmosis has occurred naturally in northern animals that never have been exposed to infestation with *Boöphilus annulatus*.

Incidentally, our naturally occurring cases also dispose definitely of a tentative opinion advanced by de Asúa and his coworkers that under natural conditions anaplasmosis follows an attack of piroplasmosis, and perhaps can occur only when an animal has been sensitized by a preceding attack of piroplasmosis. Our animals suffered from pure anaplasmosis under natural conditions.

The chief argument against the identification of these bodies with Jolly bodies is that the bodies called Anaplasma appear in the circulation of animals suffering from anaplasmosis long before any morphological changes are noticeable in the blood. If

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Jolly bodies are correctly interpreted as being remnants of red-cell nuclei, then their appearance should follow, as it actuallly does, the appearance of nucleated red cells and normoblasts, and this order should not be reversed. Anaplasma also appear in far greater numbers than Jolly bodies. An additional argument lies in the reaction of Jolly bodies and Anaplasma, respectively, to Feulgen's reagent. According to Penha,[32] Jolly bodies stain with Feulgen's reagent and Anaplasma remain unstained. Rees (in correspondence) has obtained similar results. This same reagent might be employed to determine whether the bodies produced in the blood of rabbits and guinea pigs by the injection of hemolytic poisons such as pyrogallic acid, phenylhydrazine, etc., are identical with Jolly bodies and therefore different from Anaplasma. On the basis of the available evidence, we may conclude that Anaplasma are not Jolly bodies.

The question whether Anaplasma can be considered as reaction products, resulting from the action of an ultravisible, filtrable virus, has been answered in the negative by de Kock and Quinlan.[33] These authors, working with ovine anaplasmosis, report the result of an experiment in which susceptible sheep were inoculated with (*a*) defibrinated blood, (*b*) serum, (*c*) washed corpuscles, (*d*) hemolyzed washed corpuscles and (*e*) filtrates of hemolyzed washed corpuscles passed through a Berkefeld and a membrane filter. They found that all susceptible animals injected with defibrinated blood, serum, washed corpuscles and hemolyzed washed corpuscles reacted and showed the presence of Anaplasma in the blood, whereas the animals injected with the filtrates from the Berkefeld-and-membrane filtration failed to react. On the basis of these results, they come to the conclusion that Anaplasma is not of the nature of "reaction products" set up by ultravisible, filtrable viruses. On the basis of the evidence furnished, this conclusion appears to be correct.

The results of our own experiments along this line were unsatisfactory. The first animal (26) reacted on the 57th day following inoculation with 150 cc of a mixture of serum and physiological saline, which had been passed through a No. 6 Mandler filter. The explanation suggested, at the time of the occurrence of this reaction, was that the animal had become infected accidentally through contamination of the lancet used in obtaining blood for smears. Rees subsequently showed that this explanation was tenable. In the second filtration experiment, in which one animal (30) was injected with hemolyzed red blood cells and another (31) with hemolyzed red blood cells which had

been passed through a Mandler filter, neither animal reacted, although both were susceptible as shown by subsequent inoculation. No conclusions, therefore, can be drawn from this experiment in filtration. It could not be repeated at the time because of the scarcity of animals available for experimental purposes. Whether they can be considered as reaction products resulting from an invisible, unfiltrable virus is a point that has not yet been settled.

De Asúa and coworkers report an experiment in which they took blood containing Anaplasma, hemolyzed this blood by rubbing it up with sterile sand in a sterile mortar, filtered it through sterile sand and filter paper to remove remains of the stroma of the blood-cells, which might otherwise occlude the pores of the Chamberland filter, passed the filtrate through a Chamberland LIII, and injected the last filtrate into (*a*) a cow suffering from piroplasmosis and (*b*) also into a healthy cow, in quantities of 20 and 10 cc, respectively. Neither animal reacted. They repeated the experiment a second time with similar results.

They then repeated the experiment with this difference, that they substituted a Chamberland LI for the Chamberland LIII used in the first two experiments. The reason given for this change was that, whereas the Chamberland LIII does not permit the passage of bodies the size of Anaplasma, the pores of the Chamberland LI are much larger than Anaplasma, and Anaplasma should, therefore, pass through them. Animals inoculated with the filtrate from the Chamberland LI candle also failed to react. In order to determine whether the test animals were susceptible, they were injected later with virulent blood from a known case of anaplasmosis. They reacted in due time, with the appearance of both fever and Anaplasma. The authors, therefore, come to the conclusion that the solution of the problem of etiology and the relation of the "marginal points" or Anaplasma to the disease anaplasmosis cannot be found by filtration methods and that the cause of anaplasmosis is an invisible, unfiltrable virus and that what are known as Anaplasma are the reaction products of this virus and an indication of the disease, "krankheitszeichen," rather than etiological agents of the disease. They also interpret the result of Lignières' inoculation of sheep with bovine blood containing Anaplasma and the subsequent infection of susceptible cattle following injection with the blood of sheep so inoculated, as supporting their conclusions. They argue with Wenyon that the only logical interpretation of Lignières' results, which have been confirmed in part by Sergent and coworkers and by the pres-

ent writer, is that the etiological cause of anaplasmosis is an invisible virus, which, when introduced into the blood of a sheep, remains viable in this animal without affecting it in any visible way, and is capable of reproducing the disease in susceptible bovines, when such animals are subjected to inoculation with the blood of the sheep which has been injected with bovine blood containing Anaplasma.

The conclusions drawn by these authors from their filtration experiments would have rested upon firmer ground if, in addition to injecting susceptible animals with the filtrate from the Chamberland LIII candle filtration, they had inoculated susceptible animals with the filtrate from the sand and filter paper filtration or with the blood hemolyzed by trituration with sterile sand in the sterile mortar. The results of such an experiment would have served as a control on their candle-filtration experiments, and in view of the fact that this control experiment was not performed, their conclusions cannot be accepted as adequately proven.

We may therefore conclude that (*a*) Anaplasma (*sensu stricto*) is not a phase in the life history of a member of the family Piroplasmidae; (*b*) Anaplasma is not a form of Jolly bodies; (*c*) Anaplasma is not a "reaction product" set up by an ultravisible, filtrable virus; (*d*) Anaplasma may be a "reaction product" set up by an ultravisible, unfiltrable virus; (*e*) Anaplasma may be a parasite *sui generis*.

On the basis of the evidence so far at hand, both points *d* and *e* can be maintained. Neither the question of filtration nor the question of artificial cultivation has so far received enough attention to permit of a final conclusion.

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Virginia Ladies to Meet

Mrs. M. C. Faville, secretary of the Women's Auxiliary to the Virginia State Veterinary Medical Association, advises that the Auxiliary will hold a meeting at Fredericksburg, during the meeting of the State Association, July 13-14.

HUMAN AND AVIAN TYPES OF TUBERCLE BACILLI RECOVERED FROM SWINE*

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Owing to the comparative rarity of reports concerning the finding of the human type of tuberculosis in swine, the following case appears worthy of being placed on record.

SOURCE AND HISTORY

Specimens of tuberculous lymph-glands from two garbage-fed hogs were collected by Dr. C. L. Davis, a member of the federal meat-inspection staff at Denver, Colorado, and placed at our disposal in December, 1931. The following history concerning the hogs from which the specimens were collected was furnished by Dr. Davis.

The garbage fed the hogs was collected in the city of Denver and included food from homes, restaurants, hotels, markets and sanitariums, some of which, no doubt, contained the viscera of tuberculous poultry. The feeder stated that while chickens were running with hogs in the feed-lots, he had no reason to believe his chickens were diseased. The hogs were fed garbage until they weighed about 225 pounds and were then sent to the abattoir.

Dr. Davis states that, in his experience on postmortem examination, approximately 50 per cent of this class of hogs show a cervical or mesenteric lymph-gland involvement, a combination of the two, and occasionally a carcass with more extensive lesions, or sometimes even generalized tuberculosis.

LABORATORY EXAMINATION

For the sake of convenience in handling, the specimens were given laboratory numbers, 1A and 2A, and will be so designated in the following discussion. Cultural and animal inoculation

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tests indicated that the organisms of tuberculosis isolated from the specimen of lymph-glands (1A) were of the human type, while those isolated from the other specimen of glands (2A) were of the avian type.

Specimen 1A (Human Type)

Microscopic examination of smears prepared from the specimen of hog lymph-glands revealed very few acid-fast organisms. Cultures made direct from the glands were negative.

Cultures made from guinea pigs that died with generalized tuberculosis, as a result of subcutaneous inoculation with emulsion of hog glands, showed the typical rough, dry, granular pigmented type of growth characteristic of the human type of tubercle bacilli. On various kinds of special culture media, the growth compared favorably with that of a known human strain of tuberculosis.

Animal inoculation tests: Guinea pigs inoculated subcutaneously with lymph-gland emulsion and culture emulsions died with generalized tuberculosis in from five to 20 weeks.

Rabbits inoculated subcutaneously with an emulsion of hog lymph-glands from specimen 1A developed only local lesions with a few tuberculous nodules in the lungs after a period of seven and one-half to nine months.

Rabbits inoculated subcutaneously with emulsions of tuberculous guinea-pig lesions or with young cultures, showed a nonprogressive tuberculosis consisting of a number of scattered foci in the lungs, very few foci in the kidneys, and no other visible lesions. Rabbits inoculated intravenously with a young culture showed tuberculous foci in lungs and kidneys with no generalization when autopsied four and one-half months later.

Specimen 2A (Avian Type)

Microscopic examination of smears prepared from lymph-glands showed numerous acid-fast organisms.

Cultures made direct from the specimen glands showed a smooth, moist, mushy growth, easy to emulsify, which was identical in appearance and growth characteristics on different kinds of culture media with those of a known avian strain.

Animal inoculation tests: Inoculation of chickens with cultures resulted in generalized tuberculosis in from six to ten weeks. Inoculation of guinea pigs with gland emulsion and cultures resulted in local lesions only. In some of these cases death resulted with extreme emaciation but with no visible lesions of tuberculosis.

TABLE I—*Laboratory animal inoculation tests on hog specimen 1A (human type).*

ANIMAL	METHOD OF INOCULATION	MATERIAL USED		NATURE OF DEATH	TIME AFTER INOCULATION (WEEKS)	REMARKS
G. P. 5	Subcutaneous	Emulsion of hog lymph-gland 1A		D	10	Generalized tuberculosis
G. P. 6				D	20	Generalized tuberculosis
Rabbit 7				K*	32	Few foci in lungs
Rabbit 8				K	39	Scattered foci in lungs
Rabbit 9	Subcutaneous	Spleen emulsion from G. P. 5		K*	11	Few foci in kidneys. Scattered foci in lungs
Rabbit 10				K	34	Few foci in kidneys. Scattered foci in lungs
G. P. 13	Subcutaneous	Culture 1A from G. P.	0.01 mg	D	7	Generalized tuberculosis
G. P. 14			0.01 mg	D	11	Generalized tuberculosis
Rabbit 11	Intravenous		0.1 mg	D	19	Scattered foci in lungs. Few foci in kidneys
Rabbit 12			0.01 mg	K	19	Scattered foci in lungs and kidneys
G. P. 19	Subcutaneous	Culture 1A from G. P.	1 cc	D	5	Generalized tuberculosis. Extreme emaciation
Rabbit 20			1 cc	K	11	Scattered foci in lungs. Lesion at point of inoculation

D = Died. K = Killed.
*Moribund.

TABLE II—*Laboratory animal inoculation tests on hog specimen 2A (avian type).*

ANIMAL	METHOD OF INOCULATION	MATERIAL USED		NATURE OF DEATH	TIME AFTER INOCULATION (WEEKS)	REMARKS
G. P. 1	Subcutaneous	Gland emulsion 2A		D	24	No lesions of tuberculosis. No acid-fasts in smears from spleen. Emaciation
G. P. 2				D	36	No lesions of tuberculosis. No acid-fasts in smears from spleen
Rabbit 3				D	9	No lesions of tuberculosis
Rabbit 4				K	47	Condition good. No lesions of tuberculosis
G. P. 17	Subcutaneous	Culture 2A from hog gland	0.01 mg	D	9	No lesions of tuberculosis
G. P. 18			0.01 mg	K	19	Lesions at point of inoculation. No other lesions. Condition good
Rabbit 15	Intravenous		0.1 mg	D	5	Miliary type tuberculosis in lungs. Lesions in kidneys, spleen and skin
Rabbit 16			0.01 mg	D	15	Scattered foci in lungs. Extreme emaciation. Lesions in skin and kidney
Chicken 19	Subcutaneous	Culture 2A from hog gland	1 cc	K	10	Generalized tuberculosis
Chicken 20	Body cavity		1½ cc	D	6	Generalized tuberculosis
G. P. 23	Subcutaneous		1 cc	D	4	Local lesion at point of inoculation. No other visible lesions
G. P. 24			1 cc	K	10	Local lesion at point of inoculation. No other visible lesions
Rabbit 21			1 cc	K	10	Few foci in lung. Local lesion at point of inoculation
Rabbit 22			1 cc	K	10	Few foci in lung. Local lesion at point of inoculation

Rabbits inoculated subcutaneously with an emulsion of hog lymph-glands from specimen 2A failed to develop tuberculosis, while those inoculated subcutaneously with young cultures obtained direct from the specimen of hog glands 2A developed a few scattered foci in the lungs with a local lesion at the point of inoculation.

Intravenous inoculation of rabbits with young cultures produced more extensive lesions showing a tendency towards generalization.

Tables I and II will serve to illustrate the tests on laboratory animals referred to above.

LESIONS IN CARDIAC MUSCLE OF YOUNG CALVES AS SOLE EVIDENCE OF BLACKLEG*

By C. N. DALE, *Washington, D. C.*
Pathological Division, U. S. Bureau of Animal Industry

An autopsy of a calf six months old revealed no other lesions except a black coloration and slight emphysema of the heart wall of the right auricle and the upper half of the right ventricle. A week previous, the owner had lost a two-month-old calf which was supposed to have died from digestive disorders and choke, as the calf died gasping for breath. When he performed an autopsy, he observed a blackish coloration around the heart. When the second calf died, the owner called in a veterinarian who autopsied the animal and forwarded the heart to the laboratory for examination.

Cultures of the heart muscle revealed the presence of *Clostridium chauvei.* Guinea pigs inoculated with material prepared by grinding some of the heart muscle with sterile saline solution died, showing typical lesions of blackleg. *Cl. chauvei* was recovered from the heart-blood. Microscopic examination of liver-touch preparations showed a few rods occurring singly and in pairs.

Hutyra and Marek, in their discussion of the anatomical lesions of blackleg, state that in some few cases the heart muscle shows in circumscribed areas a blackish, pale yellow discoloration. They do not mention that this lesion may occur when there are no visible lesions in other musculature of the body.

From a diagnostic standpoint the importance of recognizing or suspecting a blackish discoloration of the heart muscle of calves as a pathological manifestation of blackleg is apparent.

*Received for publication, May 22, 1933.

TWO UNUSUAL CASES OF TUBERCULOSIS IN HOGS CAUSED BY THE AVIAN TYPE OF TUBERCLE BACILLUS*

By A. B. CRAWFORD, *Bethesda, Md.*
Bureau of Animal Industry, U. S. Experiment Station

CASE 1

In February, 1932, while visiting a slaughtering establishment in Baltimore, the writer's attention was called to the retained carcass of a hog. This animal showed an enlarged, watery condition of the lymph-glands and an enlarged spleen, simulating leukemia. The spleen was of special interest, being about five times its normal volume. On its dorsal surface were scattered seven or eight slightly elevated areas, each about 1¼ to 1½ inches in diameter, purplish in color, in contrast to the brownish red of the surrounding spleen. These elevated areas resembled rosettes, being slightly roughened, with irregularly radiating striae.

A section of this spleen with one of the rosettes was placed in a sterile container and brought to this Station for examination. A smear preparation of the rosette stained with carbol-fuchsin showed several hundred acid-fast organisms per field. Cultures were made on congealed serum medium, and good growth occurred within a month.

Guinea pigs were inoculated subcutaneously with emulsions of the spleen. These animals were tested two months later with avian and mammalian tuberculins and reacted to both, but more pronouncedly to avian tuberculin. Six months after inoculation, these guinea pigs were killed but showed no lesions of tuberculosis.

Subcultures of this acid-fast were made on serum-agar. The growth on this medium was characteristic of the avian type of tubercle bacillus.

A suspension of the fourth subculture was injected into six guinea pigs and two chickens in order definitely to type this organism. The guinea pigs were killed five months after inoculation and all were free from disease.

One chicken, inoculated intravenously, died 36 days afterwards, showing a greatly enlarged liver and spleen but no tubercles (Yersin type). Hundreds of acid-fast organisms were observed in each microscopic field of smear preparations of these organs.

*Received for publication, May 23, 1933.

The second chicken, injected subcutaneously, died 95 days after inoculation. It was greatly emaciated and showed the characteristic lesions of avian tuberculosis.

CASE 2

More recently, another case of avian tubercle bacillus infection in a hog spleen was studied at this station. This specimen was submitted by Dr. G. E. Totten, inspector-in-charge, Chicago, and suspected to be of Brucella infection. The spleen was normal in appearance except for several swellings, none more than one-half inch in diameter, making slight elevations of the capsule. On palpation these areas seemed circumscribed but on incision there was little to differentiate them from the surrounding tissue, there being no necrosis or encapsulation. Smear preparations from one nodule showed a few acid-fast organisms.

A section containing one of these swellings was emulsified in normal saline solution. Six guinea pigs and two chickens were inoculated. The guinea pigs were killed two months later but showed no lesions. The chickens were tested with avian and mammalian tuberculins two months after inoculation and both reacted strongly to avian tuberculin. One of these birds showed an evident loss of weight and was killed two weeks after the tuberculin test. It showed characteristic lesions of avian tuberculosis in spleen and liver.

Missouri State Meeting

The Missouri Veterinary Medical Association will be host to the veterinarians of Illinois, at the regular annual meeting to be held in Saint Louis, July 18-19, according to an announcement made by Dr. J. D. Ray, Secretary. The meeting will be held at the Melbourne Hotel, Grand Ave. and Lindell Blvd., "where you can get a room and bath for a dollar and a half."

A very interesting program has been arranged, including addresses by four well-known members of the medical profession, on rabies, tuberculosis, undulant fever and public-health work. There will be a banquet, with addresses by Dr. N. F. Williams, president of the A. V. M. A., and four ex-presidents of the national organization. Lots of free entertainment will be provided for the ladies and children.

Come to Chicago—August 14-15-16-17-18

HANDBUCH DER CHEMOTHERAPIE. ERSTER TEIL, METALLFREIE ORGANISCHE VERBINDUNGEN. (Manual of Chemotherapy. Part I, Metal-Free Organic Compounds.) Victor Fischl and Hans Schlossberger. viii + 357 pages. Fischer's Medical Bookbinders, Leipzig, 1932. Price, M. 34.

This is a monograph of 357 pages devoted to the chemical constitution and therapeutic use of a large number of metal-free organic compounds. Only the compounds of known and established usage are included. To meet these requirements the authors reviewed and examined a vast amount of literature on the subject and have brought together the ideas and findings of the chemist and medical man concerning the compounds discussed. They have attempted to group and classify the different compounds with the result that it facilitates references to similar substances. Chloroform, carbon tetrachlorid, ethyl chlorid, hexachlorathan and tetrachlorethylene are grouped as acyclic chlorin compounds. The other groups include the unsaturated fatty acids, benzol and naphthaline derivatives, animoacids, quinoline derivatives, quinin and its derivatives, emetin and its derivatives, plant glucosids and alkaloids, acridin derivatives, the organic dyes, urea derivatives and some of the antiserums. In many cases they have given a brief but fairly complete history and discussion of the compounds described as well as the chemical, pharmacological, toxological and clinical application of them. The monograph should prove of great value to therapeutists and internists.

H. C. H. K.

DIE TIERE IM CHEMISCHEN KRIEGE. (Animals in Chemical Warfare.) Prof. C. E. Richters. 40 illustrations in the text and 5 colored plates. Richard Schoetz, Berlin, 1932. Paper, M. 9,60; cloth, M. 11,40.

This is an exceedingly interesting monograph dealing with the effects of various chemical compounds, particularly gases on animals. These, of course, were employed in the late war. Although interest in such effect probably is limited, nevertheless

those veterinarians who are in the Army or in the Reserve Corps, should certainly be familiar with the wealth of knowledge in this book.

On page 21 there is a table giving the names of the gas compounds with their military designations, their chemical formulae, physical properties, more or less dense than air, the boiling points, and whether each compound is decomposed by the presence of water vapor. The principal gases, of course, were chlorin, mustard gas (dichlordiethyl-sulphid), and phosphene. Excellent illustrations appear in the text of the effect these compounds have on animals coming in contact with them. Descriptions of various treatments used and methods of prevention are given at some length. The reviewer does not believe that anywhere else has there been collected anything like the information obtained between these covers in respect to chemical warfare and its effect upon the animal population. Dr. R. A. Gortner, of the Department of Bio-Chemistry, University of Minnesota, was asked to look over the chemistry given in the monograph, and he states that as far as he can see the chemistry is all right, but limited to a consideration of the different chemical compounds which can be used under war conditions in an "offensive attack." The colored plates at the end of the book vividly illustrate certain of the pathology produced by these compounds. This monograph should be of interest to many.

C. P. F.

PUBLICATIONS RECEIVED

Veterinary Director General for the year ending March 31, 1932, Report of the. George Hilton. (Canada Dept. of Agri., Ottawa, Ont., 1933. pp. 53.)

Pennsylvania Society for the Prevention of Cruelty to Animals, Sixty-fifth Annual Report of the. (922-924 N. Broad St., Philadelphia, Pa., 1932. pp. 24.)

Portland, Oregon, Bureau of Health Report for 1932. R. E. Riley, Commissioner. (Portland, Ore., 1932. pp. 26.)

The Sheep Blowfly Problem in Australia. Report No. 1 by the Joint Blowfly Committee. (Pamph. 37, Coun. for Sci. & Ind. Res., Commonwealth of Australia. Melbourne, Australia, 1933. pp. 142. Illus.)

Measures to Control Colics (Stoppage of Bowels) in Horses, Mules and Donkeys in Saint Croix, Virgin Islands. G. A. Roberts. (Virgin Islands Agri. Sta., U. S. Dept. of Interior, Christiansted, Saint Croix, Virgin Islands, 1933. pp. 4.)

Worms in Hogs. (Circ. 140, Montana Vet. Res. Lab., Montana Agri. Exp. Sta., Bozeman, Mont., 1933. pp. 8. Illus.)

Intestinal Worms in Poultry. (Circ. 141, Montana Vet. Res. Lab., Montana Agri. Exp. Sta., Bozeman, Mont., 1933. pp. 6. Illus.)

Intestinal Worms in Sheep. (Circ. 142, Montana Vet. Res. Lab., Montana Agri. Exp. Sta., Bozeman, Mont., 1933. pp. 8. Illus.)

The Register of Veterinary Surgeons. (Royal College of Veterinary Surgeons, London, England, 1933. pp. 309.)

A Histochemical Study by Microincineration of the Inclusion Body of Fowl-Pox. W. B. C. Danks. Amer. Jour. Path., viii (1932), 6, pp. 711-716. Illus.

The Blood Picture of Pigs Kept Under Conditions Favorable to the Production and to the Prevention of So-Called "Anemia of Suckling Pigs." H. C. H. Kernkamp. (Tech. Bul. 86. Univ. of Minn. Agr. Exp. Sta., Dec., 1932. University Farm, Saint Paul, Minn. pp. 32.)

New South Wales Veterinary Research Report No. 6, Part III (Dec., 1931). H. R. Seddon, Director. (New South Wales Dept. of Agr., 1933. Glenfield, N. S. W., Australia. pp. 95-136. Illus.)

"Tick Borne Fever" (A Hitherto Undescribed Disease of Sheep). W. S. Gordon, A. Brownlee, D. R. Wilson, and J. MacLeod. Reprint from *Jour. Comp. Path. & Therap.*, xlv (1932), pt. 4, pp. 301-312.

Diseases Affecting Moose. R. Fenstermacher. (Bul. 294. Univ. of Minn. Agr. Exp. Sta., Jan., 1933. University Farm, Saint Paul, Minn. pp. 20. Illus.)

Water Hemlock (*Cicuta maculata* L.) Poisoning in Swine. Louis V. Skidmore. Reprint from *Vet. Jour.*, lxxxix (1933), 2, pp. 76 80. Illus.

Pine: A Disease Affecting Sheep and Young Cattle. J. Russell Greig and Henry Dryerre, in collaboration with W. Godden, A. Crichton, and W. G. Ogg. Reprint from *Vet. Jour.*, lxxxix (1933), 3, pp. 99-110. Illus.

Pregnancy Disease in Ewes. F. B. Hadley. (Spec. Circ. Coll. of Agr., Univ. of Wis., April, 1933. Madison, Wis. pp. 2.)

Comparative Histology and Cytology of Infectious Diseases Caused by Filtrable Viruses. O. Seifried. Vet. Ext. Quar., Univ. of Pa. Bul., xxxiii (1933), 50, pp. 23. Illus.

The Rocky Mountain Wood Tick. R. A. Cooley. (Bul. 268, Mont. Agri. Exp. Sta., Bozeman, Mont., 1932. pp. 58. Illus.)

Avian Tuberculosis in Cattle in Great Britain. F. C. Minett. Reprint from *Jour. Comp. Path. & Therap.*, xlv (1932), pt. 4, pp. 317-330.

A Diphtheroid Organism Showing Acid-Fast Properties in Milk Smears. G. Martinaglia. Reprint from *Jour. S. African Vet. Med. Asso.*, iii (1932), 4, pp. 4. Illus.

Swine Erysipelas. T. W. Munce. Reprint from *Allied Vet.*, iv (1933), 2, pp. 5-10. Illus.

The No-Lesion Case Problem in the Tuberculosis Eradication Campaign. W. A. Hagan. Reprint from *Corn. Vet.*, xxi (1931), 2, pp. 163-176.

Soremouth (Contagious Ecthyma) in Sheep and Goats. H. Schmidt and W. T. Hardy. (Bul. 457, Tex. Agr. Exp. Sta., College Station, Tex., 1932. pp. 22.)

Blood Volume Determinations in Cattle. W. T. Miller. Reprint from *Corn. Vet.*, xxii (1932), 4, pp. 320-332.

The Transmission of Tuberculosis by Contact from Infected to Healthy Guinea-Pigs, with Reference to Some Recent Work on B.C.G. Vaccine. T. M. Doyle. Reprint from *Vet. Jour.*, lxxxix (1933), 1, pp. 30-34.

Six Years' Experience with a Herd Experimentally Infected with Johne's Disease. W. A. Hagan and Alexander Zeissig. Reprint from *Corn. Vet.*, xxiii (1933), 1, pp. 1-15.

THE PRESENCE OF TUBERCLE BACILLI IN HENS' EGGS. S. Lichtenstein. Abst. Arch. Path., xv (1933), 2, p. 298.

Live and virulent tubercle bacilli were found in 0.38 per cent of 525 market eggs, and in 0.46 per cent of 216 eggs from hens living in tuberculous surroundings. In eggs artificially infected with avian bacilli, tubercle bacilli could be isolated in pure culture up to the 167th day, and they were demonstrable morphologically up to the 198th day.

CASEOUS LYMPHADENITIS: INGESTION AS A METHOD OF INFECTION WITH THE PREISZ-NOCARD BACILLUS. THE GROWTH OF THE PREISZ-NOCARD BACILLUS IN SHEEP FECES. H. R. Carne, Abst. Vet. Bul., iii (1933), 1, p. 14.

If lesions connected with the alimentary tract can be accepted as a criterion of the method of infection, natural infection of sheep by ingestion is uncommon. In experimental infections by this route, lesions are usually confined to lymphatic glands draining from the buccal cavity, as conditions in the caudal portion of the bowel are apparently not suitable for the penetration and production of lesions by *Corynebacterium pseudotuberculosis ovis*. Experimental evidence showed the gastric juice of sheep to have a variable action on the organism, but attempts to infect *per rectum* were unsuccessful. Sheep fed on nitrogenous food excrete feces which, when sterilized, support the growth of *C. pseudotuberculosis ovis*. In unsterile conditions the organism is probably overgrown by others in feces, but until it has been proved to the contrary, precautions must be taken against feces as a possible vehicle of infection.

THE EFFECT OF BLOOD LOSS UPON THE RESISTANCE OF CHICKENS TO VARIABLE DEGREES OF PARASITISM. Dale A. Porter and James E. Acker. Amer. Jour. Hyg., xv (1933), 1, p. 252.

Two experiments on a total of 300 White Leghorn chickens were conducted in this investigation. The criteria of determin-

ing whether the resistance of the chickens to the parasite was affected were the numbers of nematodes (degree of infestation) and the lengths of the worms (rate of growth) that occurred in the various lots of chickens under comparison. The results of the experiments indicate that repeated loss of blood may lower the resistance of chickens to the intestinal nematode *Ascaris lineata*. When large numbers of eggs (500 to 300) were fed, three of the four lots bled showed significantly longer worms than did the control lots of chickens (not bled). While several comparisons did not result in significant differences, the worms from the bled lots of chickens were longer than those from the controls in nine of the ten groups in which comparisons were made. The lowered resistance was thought to have been due to a physiological reaction in which the growth-inhibiting mechanism was affected.

RESPIRATORY METABOLISM AND TISSUE RESPIRATION IN EXPERIMENTAL ANAEROBIC INFECTION. G. Schmid. Abst. Arch. Path., xv (1933), 1, p. 137.

One set of experiments consisted in the determination of the oxygen consumption, carbon dioxid production, and the respiratory quotient of guinea pigs infected with the organisms of malignant edema, the results being compared with those of the same animals before infection. Similar determinations were made on a small series of animals infected with aerobic organisms of fowl cholera. The figures are so variable and inconsistent as not to warrant any conclusions. Tissue respiration was studied by the method of decolorization of methylene blue solution. The muscles of animals infected with malignant edema decolorized the solution more slowly than the muscles of animals infected with fowl cholera.

COCCIDIOSIS OF THE LIVER OF RABBITS. I. Experimental study of the excystation of oöcysts of *Eimeria steidae*. Hans Smetana. Arch. Path., xv (1933), 2, p. 175.

The excystation of oöcysts of *Eimeria steidae* is induced by the enzymatic action of the pancreatic juice; it is then completed by the spontaneous movements of the sporozoites. The specific enzyme concerned is trypsin-kinase. This can be demonstrated by: (1) the inertness of enzymes other than trypsin as regards influence on excystation; (2) the certain parallelism between the tryptic activity of the medium on casein and the liberating effect of the medium on sporozoites of oöcysts; and (3) the influ-

ence of activation by enterokinase. The optimum hydrogen-ion concentration for excystation is between pH 7 and pH 8. Excystation takes place at 37°C. and cannot be effected at room temperature. The length of time required to induce excystation *in vitro* varies considerably; the minimum is about 60 minutes and the optimum between five and seven hours. The response to the action of trypsin of oöcysts of *Eimeria steidae* obtained from lesions of the liver differs somewhat from that of oöcysts collected from the feces. Exposure of oöcysts to gastric digestion prior to intestinal digestion does not influence the process of excystation.

TRANSMISSION OF CHICKEN LEUKOSIS TO CHICKS. J. Engelbreth-Holm and A. Rothe Meyer. Abst. Arch. Path., xv (1933), 2, p. 286.

Chicken erythroleukosis can be successfully transmitted to chicks a few days old. The disease shows in them more marked changes and a faster course than in adult animals. The transmission was successful in all cases, even when injection of the same material into adult chickens did not produce the disease.

THE EFFECT OF VARIOUS THERAPEUTIC AGENTS ON BRUCELLA ABORTUS INFECTION IN GUINEA PIGS. C. M. Carpenter and R. A. Boak. Abst. Jour. Bact., xxv (1933), 1, p. 88.

Seventy-five guinea pigs were injected subcutaneously with a culture isolated from man's blood, and fifty with a strain isolated from an abscessed epididymis of a bull. Twenty were kept for controls, while groups of from ten to twenty were treated by one of the following methods: (1) acriflavine intraperitoneally, (2) methyl violet *per os*, (3) thionin *per os*, (4) immune serum intraperitoneally, (5) feeding autogenous vaccine, (6) injecting autogenous vaccine subcutaneously, (7) deep x-ray therapy and (8) artificially induced fevers. The dyes and the subcutaneous injection of heat-killed vaccine failed to control the infection. Improvement was noted following the use of immune serum, feeding vaccine, and artificially induced fevers.

A NEW METHOD OF SECURING CLEAR SERUM FROM THE MILK OF COWS AND GOATS FOR AGGLUTINATION TESTS. Ivan C. Hall and Robert Learmonth. Jour. Inf. Dis., lii (1933), 1, p. 27.

Ether, benzene, carbon tetrachlorid, chloroform, carbon disulphid and tetrachlorethane were added as fat solvents to milk

drawn for agglutination purposes. Carbon tetrachlorid and chloroform were found to be satisfactory. Corked tubes were prepared, each containing a few cubic centimeters of the solvent and a small amount of rennet extract. The sample is drawn directly from the animal into the tube. Each tube is shaken a few minutes to dissolve the fat. The tubes then are placed in the incubator at 37° C. for one hour to coagulate the casein. They are then centrifuged for 15 minutes at 2,000 revolutions per minute. The milk-serum collects in a deep clear layer at the top, a curd in the center, and the solvent at the bottom. Such milk-serums can be preserved indefinitely without bacterial growth. There is no interference with the agglutination test.

FURTHER ATTEMPTS AT EXPERIMENTAL INFECTION OF MAN WITH A BOVINE STRAIN OF BRUCELLA ABORTUS. P. Morales-Otero. Jour. Inf. Dis., lii (1933), 1, p. 54.

A family consisting of three members volunteered to ingest, for six weeks, the infected milk of a cow suffering from infection with *Br. abortus*. At the end of this period, none of the members had at any time shown any symptoms of the disease and their blood cultures and their blood agglutination tests remained persistently negative for *Br. abortus*. Two other volunteers inoculated, one through normal and the other through abraded skin with the strain isolated from the infected cow's milk, presented no symptoms of the disease and their blood-cultures were persistently negative for *Br. abortus*. A transitory reaction (1:40 dilution) was observed in the latter cases during the third and fourth weeks of the experiment.

THE APPARENT CAUSE OF AN INFECTIOUS ENTERITIS OF VERY YOUNG LAMBS. Hilda Hempl Heller. Jour. Bact., xxv (1933), 1, p. 91.

There exists in California and elsewhere a fatal epizoötic enteritis of very young lambs, that is associated with lambing in sheds and occurs in widely separated fulminating outbreaks. Diarrhea may or may not occur; death may supervene at the age of one or two days; there is a severe toxemia and there are frequently nervous symptoms. Characteristic lesions are: severe hemorrhagic enteritis isolated in stretches but not in patches of the small intestine, necrosis of the intestinal epithelium, infiltration of the mesenteric lymph-nodes, injection of the adrenals, congestion and atelectasis of large, circumscribed portions of the

lungs, and engorgement of the cerebral and other blood-vessels. The tissues and blood of the lambs frequently are sterile. Filtered extracts of the intestinal contents contain a powerful poison which resists boiling and is not neutralized by botulinus antitoxins of types A, B, C or D; 0.25 cc of such extract may kill an injected mouse in 2.5 hours. Mixed cultures and their filtrates from a large number of lambs contained this poison which, on intraperitoneal injection, prostrated guinea pigs and caused death by respiratory failure in a few hours. Lesions of these guinea pigs resembled those of the lambs. A long series of experiments in which lambs were fed treated intestinal content and cultures showed that it was highly probable that an anaerobic organism causes this disease. Some of the colonies of colon bacillus from the intestine produced a poison of the identical effect as that of the intestinal extract and the mixed cultures; 4 cc of filtrate from one colony culture killed a guinea pig in 2.5 hours. The loss of pathogenicity of the colon bacillus strains was phenomenally rapid; their colony variants were numerous. This enteritis differs in lesions from English "lamb dysentery"; it resembles closely iso-colon bacillus of calves (Christiansen). It partakes of the nature of food-poisoning and of infection. It would seem to be caused by the formation of a poison in the milk in the intestines of lambs, by a virulent stage of a colon bacillus that is highly variable. Death is usually due to absorbed poison; septicemia, however, may occur.

OPTIMAL PROPORTIONS IN AGGLUTINATION: With reference to the antigenic analysis of the Brucella group of organisms. A. A. Miles. Brit. Jour. Exp. Path., xiv (1933), 1, p. 43.

The velocity of agglutination in a constant volume of fluid is at a maximum when the ratio of the concentrations of bacteria and immune serum has one of two values, depending on the method of titration. These values are constant, and bear a constant relationship to one another for a given antiserum and antigen. One ratio is determined by titration: antigen against a constant antiserum concentration, the other by titrating antiserum against a constant antigen concentration. The significance of and the relations between the two ratios are discussed. Secondary optimal ratios of as yet unknown significance in the constant antigen titrations are described. Representative smooth strains of *Br. abortus* (bovine variety) and *Br. abortus* (porcine variety) are distinguishable from *Br. melitensis*. By use of the

optimal proportions technic the abortus varieties are indistinguishable from one another. The limitations of the method applied to antigenic analysis of bacteria are discussed.

BIOLOGICAL STUDIES OF THE TUBERCLE BACILLUS. II. A new conception of the pathology of experimental avian tuberculosis with special reference to the disease produced by dissociated variants. W. A. Winn and S. A. Petroff. Jour. Exp. Med., lvii (1933), 2, p. 239.

Four variants, S, F S, R and Ch, dissociated from an avian tubercle bacillus, are described in detail by the authors. They have different physical and chemical properties. The leucocytic response in S and F S is of the acute type, while that produced by the R and Ch variants is indicative of a chronic, healing tuberculosis. The tubercle formed by S is of an acute toxic type, the F S more of a foreign-body type, and that of R and Ch relatively benign. The S variant is by far the most virulent and is closely followed by the F S type. The R and Ch variants are comparatively avirulent.

A SUCCESSFUL METHOD FOR THE TREATMENT OF STRYCHNIN POISONING; REPORT OF ELEVEN CASES. G. F. Kempf, J. T. C. McCallum and L. G. Zerfas. Jour. Amer. Med Asso., c (1933), 8, p. 548.

Sodium amytal and sodium pentobarbital given intravenously are definite life-saving measures in strychnin poisoning in man. The drug is given in sufficient doses (repeated if necessary) to stop convulsions. Gastric lavage is unnecessary.

Free Motor Travel Service

Motorists to the A. V. M. A. convention who have not mapped out their routes of travel will find a helpful ally in the Conoco Travel Bureau, Denver, Colo. The Bureau offers free travel service, and will send on request a set of state maps, showing the best and most direct routes to the convention. The types of roads and road conditions are indicated, and lists of hotels and cottage camps included. The Bureau will also supply literature descriptive of places of interest along the way.

Come to Chicago—August 14-15-16-17-18

Regular Army

Second Lieutenant W. W. Bertz, who was recently ordered assigned to the Port of Embarkation, Brooklyn, N. Y., has now been assigned to Fort Bliss, Tex., for duty. Upon entering on his new assignment, Lieutenant Bertz is relieved from further duty at the Army Veterinary School, Army Medical Center, Washington, D. C., and from temporary duty at the Medical Field Service School, Carlisle Barracks, Pa., effective upon the completion of his present course of instruction at the Medical Field Service School.

Second Lieutenant Ralph W. Mohri is relieved from duty at Fort Riley, Kan., effective on or about August 29, 1933, and will then report to the commandant, the Cavalry School, for duty as a student in the 1933-34 troop officers' course. The promotion of Lieutenant Mohri to the grade of First Lieutenant, to rank from April 23, 1933, also is announced.

Veterinary Reserve Corps

New Acceptances

Cameron, Hugh Stuart..2nd Lt...515 Dryden Rd., Ithaca, N. Y.
Marsh, Edward Thornton..Capt...312 Federal Bldg., St. Joseph, Mo.

Promotions

To

Bolenbaker, Roger Fraleigh..Capt. Box 74, 403 E. Q. St., Wilmington, Calif.
Brandly, Carl Alfred..1st Lt.....1026 Kearney St., Manhattan, Kan.
Chastain, Walter Ralph..1st Lt...408 State Office Bldg., Columbia, S. C.

Colonel and Mrs. Hill Entertained

Dr. and Mrs. N. S. Mayo, of Highland Park, Ill., entertained Col. and Mrs. Wm. Proctor Hill, of Fort Meade, Md., and a number of their old-time friends, Sunday evening, June 25.

Come to Chicago—August 14-15-16-17-18

COMMENCEMENTS

ONTARIO VETERINARY COLLEGE

Seventeen candidates received the degree of Bachelor of Veterinary Science at the Convocation of the University of Toronto, held on May 5, 1933. They are:

A. F. Bain
H. T. Batt
R. J. Bilyea
M. O. Bingeman
W. J. Boyd
M. E. Epperson
H. A. Fraser
R. T. Gilbert
C. W. Gollehon
V. K. Jensen
C. H. Kable, Jr.
D. L. MacLean
R. E. Nichols
O. S. Nordland
G. Rankin
F. R. Rasmussen
E. A. Willick

Honors were awarded to members of the graduating class as follows:

Honor Standing—Written Examinations

Gold Medal for highest aggregate standing—H. T. Batt, of Guelph, Ont.

Second Prize—A. F. Bain, of Guelph, Ont., and H. A. Fraser, of Queenstown, British Guiana.

Third Prize—O. S. Nordland, of Winnipeg, Man.

Honorable Mention—R. E. Nichols, of Utica, N. Y.

Andrew Smith Memorial Medal

H. A. Fraser, of Queenstown, British Guiana.

Helen Duncan McGilvray Prize

O. S. Nordland, of Winnipeg, Man.

Canadian Army Veterinary Corps Prize

H. T. Batt, of Guelph, Ont.

Bacteriology

Special Prize, A. F. Bain, of Guelph, Ont.

UNIVERSITY OF GEORGIA

The 133rd annual commencement exercises of the University of Georgia were held June 5, 1933. At these exercises the following senior students of the Division of Veterinary Medicine received the degree of Doctor of Veterinary Medicine:

John William Carlton
Tim B. Clower
William L. Cooley
James C. Gibson
John A. Giddens
Conrad R. Hinson
Samuel T. Johnson
Thomas J. Jones
Henry E. Martin
Lewis C. Rossman
Claude O. Wilder

As announced in the May issue of the JOURNAL, the veterinary course at the University of Georgia is to be discontinued. The

total number of veterinary graduates of the institution (1921-1933) now stands at 70.

KANSAS STATE COLLEGE

The 70th annual commencement exercises of the Kansas State College were held at Manhattan on June 1, 1933. In the Division of Veterinary Medicine, 40 graduates received the degree, Doctor of Veterinary Medicine. They are:

Linden Moore Alcorn
Gayle Derwood Allen
Olin Alvin Anderson
William Joseph Angerer
*Loren Cleatus Blackburn
*Thomas Lenord Bond
Paul Jacob Brandly
Allen Vincent Brunke
*Wilmer I. Conger
*Grant Fuller Cottrell
*Ray Curry
*Elmer Fred Finke
Lendall Kiple Firth
Paul Carl Geilenfeldt
*Robert Henry Gump
John Lowell Hakl
Raymond William Hayes
Arthur James Hoffman
*Claude Hudson
*Harlow Kenyon Hudson
William Francis Irwin
*Conley Gordon Isenberg
*Richard Hulett Jurden
*Manuel Charles Kastner
William Hautecoyne Lindley
Clifford Ladell McGinnis
*Velmer Wayne McGinnis
Robert Tulloss McLean
Everil Dwain Merkley
*Joseph Fedelis Nieberding
*Eugene Way Peck
Charles Deets Pickett
*Charles Joseph Prchal
*Jake Louis Reineccius
Alexander Stephen Robertson
*Ralph Franklin Shaner
Hubert Leslie Smith
*William Birchard Snodgrass
*Thomas Marion Thompson
Richard Duncan Turk

Twenty of the graduating class (indicated by *) received commissions as second lieutenants in the Officers' Reserve Corps of the United States Army.

Richard Duncan Turk was awarded high honors, and Harlow Kenyon Hudson, William Hautecoyne Lindley and Eugene Way Peck were awarded honors in the Division of Veterinary Medicine.

Dr. Arthur Esco Bate (K.S.C. '19) of Witchita, Kan., received the degree Master of Science. His major subject was Bacteriology and the title of his thesis was "A Study of the Coliform Group of Bacteria from the Intestines of Diseased Chickens."

STATE COLLEGE OF WASHINGTON

The annual commencement exercises of the State College of Washington were held June 5, 1933. The following graduates received the degrees of Doctor of Veterinary Medicine and Bachelor of Science:

Balfour Bigelow (*cum laude*)
William Brimer
Roy Carey

Wallace Flynn
Blas Garcia
Gordon Hambrook
Arthur Jerstad
(*magna cum laude*)
Lyle Moss
Allen M. Northrup
(*summa cum laude*)
Catherine Elizabeth Roberts
(*cum laude*)
Marvin Thorpe
(*cum laude*)

The presentation of the candidates was made by Dean E. E. Wegner, of the College of Veterinary Medicine.

Seven of the eleven graduates took the Washington State Board examinations immediately following graduation.

COLORADO AGRICULTURAL COLLEGE

The 1933 commencement exercises of the Colorado Agricultural College were held on June 8. The commencement address, "Answering the Call of Life," was delivered by Dr. Bruce Curry, of the Union Theological Seminary, New York City.

The following 21 students were graduated from the Division of Veterinary Medicine, according to Dean George H. Glover:

Wilfred S. Bentham
Fred W. Brand
Robert E. Dunn
Phil H. Graves
William L. Hatcher
Lester Heath
Harry E. Kingman, Jr.
Charles Koberg
Leland D. Leonard
Marley C. McFadden
Ross E. Meader, Jr.
P. S. Millard
Manuel Navarro
Norton A. Orr
Paul G. Palmer
G. A. Ruggles
E. J. Siemer
William J. Snodderly
S. N. Studer
J. L. Sullivan
Rex E. Tinsman

At the end of the first semester 1932-33, Otho Jacobson received his degree in Veterinary Medicine.

The 1933 scholarship honor in the Division of Veterinary Medicine went to Lester Heath. The Alpha Psi cup for the best grades in the sophomore year went to Lorenz St. Clair.

MICHIGAN STATE COLLEGE

At the seventy-fifth annual commencement exercises of the Michigan State College, June 12, 1933, the following graduates in the Division of Veterinary Science received the degree, Doctor of Veterinary Medicine, according to Dean Ward Giltner:

Max Abraham
Fred Gasow
Charles Grey
Mary McCollum
Charles Morrill
Kenneth Van Nocker

Everett Pringle received the degree of Doctor of Veterinary Medicine at the conclusion of the fall term in 1932.

Charles Morrill won the Michigan State Veterinary Medical Association Prize of $25, which is pledged by the Association and awarded each year by the veterinary faculty to the senior veterinary student doing the best work in his course. Mr. Morrill also received one of the M. S. C. Scholarships. These awards, consisting of $100 each, represent the income from $4,000, which has been set aside by the State Board of Agriculture. They are given each year to the man and the woman student, respectively, who, at the close of their junior year, shall have shown the best scholastic record considered in its broadest sense, regardless of the course pursued.

David Clark received the Veterinary Prize of $25, which is awarded at the opening of the fall term to the veterinary sophomore who, during his freshman year, has made the best scholastic record of the class.

The seniors took the Michigan State Board examinations, June 5 and 6.

IOWA STATE COLLEGE

Commencement exercises at Iowa State College were held June 12, 1933. On that occasion the degree of Doctor of Veterinary Medicine was conferred on 39 graduates as follows:

*William H. Ahlers
Andrew C. P. Anderson
Lee K. Bailey
Theodore F. Bartley
Charles E. Bild
Chester A. Brecher
Guy C. Brown
John B. Champlin
*John Chenoweth, Jr.
*George Leonard Collins
Nels F. Christensen
Harold B. Dailey
Robert M. Dissinger
Walter F. Ellgen
Robert F. Estes
Merrill F. Frevert
Raymond R. Griffel
W. Lewis Harter, Jr.
James P. Hoben
Martin Johnson
Harry W. Johnson
Donald O. Kitchen
*Emil V. Larson
Mitchell Luinstra
Howard C. Maxey
Leland D. Mechem
Benjamin Meerdink
John D. Morton
Ivan Oelrich
Paul L. Piercy
*Benhamin S. Pomeroy
M. D. Radloff
Kenneth L. Ritchie
Glenn G. Seney
Charles J. Sinek
Lloyd C. Tekse
Mervin I. Thiele
F. Arnold Todd
G. Harold Todd

Five of the graduates (indicated by *) are sons of veterinarians, according to Dean C. H. Stange, of the Division of Veterinary Medicine.

George Leonard Collins was honor student in the Division of Veterinary Medicine and thereby won the George Judisch Prize, consisting of membership in the A. V. M. A. and subscription to the JOURNAL, for five years, valued at $25. He won also the first G. G. Graham Prize for the best work in clinical medicine. W. Lewis Harter, Jr., won the second prize.

Iowa State College this year celebrated its 75th anniversary.

OHIO STATE UNIVERSITY

The fifty-sixth annual commencement exercises of the Ohio State University were held on June 12, 1933. The commencement address was delivered by Gaius Glenn Atkins, D. D., L. H. D., of Auburn Theological Seminary.

The College of Veterinary Medicine presented 36 candidates for the degree, Doctor of Veterinary Medicine, as follows:

James William Armstrong
David Henry Bachtel
Maurice Frank Belden
Thomas Alfred Berry
Melvin James Bramley
Bertram Novell Cameron
Max Sibley Coates
J. Lavere Davidson
James Bruce Davison
Howard James Dixon
Robert Nixon Earhart
Matthew J. Eggert
Frank Joseph Elberson
Walter Gilmore Galloway
Charles Albert Goldsmith
Gilbert Neal Haigler
William Richard Henson
James Scott Landis
Arthur Albert Maike
Forrest McClead
Graydon Smith McKee
Carl Van McKenzie
John Leslie McKitrick
Byron Russell Moore
Rudolph Oscar Omdalen
Edwin Robert Pierce
Harry Ben Roberts, Jr.
James Ewing Scatterday
Marion Woolever Scothorn
Kenneth Frost Smith
Aaron Isaac Stern
Joseph Henry Straker
Newton Bridgewater Tennille
Edwin King Treat
Martin Lyon Tuttle
Paul T. White

The degree, Master of Science in Veterinary Medicine, was received by the following candidates:

Perry H. Blickenstaff (Wash. '23)
George Frederick Delaplane (O. S. U. '32)
Guilford Smith Elwood ((O. S. U. '32)
George Dewey Jelen (O. S. U. '23)
Emmett Karl LeDune (O. S. U. '29)
Theodore Earl Nichols (O. S. U. '21)

In the absence of President George W. Rightmire, due to illness, Dr. William McPherson, Dean of the Graduate School, conferred the degrees following the formal presentation of the candidates. The diplomas were awarded by Dean Oscar V. Brumley.

ALABAMA POLYTECHNIC INSTITUTE

The following graduates received the degree of Doctor of Veterinary Medicine from the College of Veterinary Medicine of Alabama Polytechnic Institute at the commencement exercises on June 16, 1933:

Percival G. Barber
Lee M. Becton
Hanceford W. Hayes
Eugene B. Hubster
Urton Munn
Everette A. Price
Haskel B. Rawls

Four others will be graduated at the end of the summer school according to Dr. C. A. Cary, Dean of the College.

A. AND M. COLLEGE OF TEXAS

Dr. Mark Francis, Dean of the School of Veterinary Medicine of the A. and M. College of Texas, announces the following graduates who received their degrees in Veterinary Medicine on June 3, 1933:

S. E. Bunton, Jr.
J. W. Dollahite
J. B. Mims
W. A. Wilson

CORNELL UNIVERSITY

At the sixty-fifth annual commencement exercises of Cornell University, on June 19, 1933, 23 graduates were presented by Dean W. A. Hagan, of the New York State Veterinary College, to receive the degree, Doctor of Veterinary Medicine. They are:

Reynold Leslie Bean
Harold LaRue Brown
Fred Forbes Bushnell
Fay Beckstead Carragher
Arthur Gordon Danks
Philip Laird Dunnet
Arthur James Fallon
Edward Clark George
Helen Baylin Goldhaft
David I. Hollenbeck
Frederick James Hoyt
William Joseph LaFrance
Michael Paul Lawrence
Nelson Richard Liner
Edward Stanley Morkis
Richard Calvin Olmstead
Henry Elmer O'Neil
Frederick William Schutz
Richard Martin Sears
Morris Harry Shapiro
Webster Eugene Speer
Paul Herbert Archibald Wilkes
Herbert Wohnsiedler

Charles Edward Fletcher was granted the degree, Doctor of Veterinary Medicine, at the close of the first term in February.

The following prizes were awarded for the academic year 1932-33:

The Horace K. White Prizes (Meritorious Students):
First Prize ..... Arthur Gordon Danks
Second Prize ..... Henry Elmer O'Neil

The Jane Miller Prizes (Veterinary Physiology):
First Prize ..... Ellis Pierson Leonard
Second Prize ..... Osman Babson

The James Gordon Bennett Prize (Surgical Clinics): .....
..... Harold LaRue Brown

The Anne Besse Prize (Veterinary Medicine)..... Henry Elmer O'Neil

The Charles Gross Bondy Prizes (Small-Animal Clinics):
First Prize ..... Arthur James Fallon
Second Prize ..... Arthur Gordon Danks

The Merry Prize (Anatomy):
First Prize ..... Esther Teich
Second Prize ..... equally divided between
..... John Chester Sweatman and Michael Joseph Donnahue

A Peculiar Find

What may be said to be a peculiar find of skulls of horses is reported in the *Veterinary Journal* (London) for May, 1933.

The discovery was made in an old house in Bungay, Suffolk, where, it appears, the floors are underlain with the skulls of horses. Several were recovered when carpenters had occasion to repair the floor of the dining room some months ago, and in February, 1933, eight were recovered when two boards were removed from the floor of another room. The report says that there appeared to be skulls as far as one could reach. Each head rested directly on the earth, with the incisor teeth supported on a small block of oak or stone wedged so firmly between the ground and the flooring that the carpenter had to knock it away with a hammer. The heads were placed with great regularity.

The house is old, and is supposed to have been standing before 1688, when Bungay was destroyed by fire almost completely. It is below the level of the present roadway by some eighteen inches or two feet. The flooring, of red pine, is in good condition. The joists are of oak, some of which have rotted away.

Collie 'Phones for Help

When a Chicago housewife returned home after taking her husband to the hospital, she fainted. There was no one in the house but Kayo, the family collie. He knocked over the telephone and barked into the mouthpiece until the operator called the nearest police station. Two policemen soon arrived and secured medical aid for Kayo's mistress.

KANSAS CITY VETERINARY ASSOCIATION

The Kansas City Veterinary Association has been holding exceptionally interesting meetings during the last few months. Dr. Ashe Lockhart is chairman of the Program Committee.

Fifty members and visitors attended the meeting on Tuesday evening, February 21, 1933, at the Baltimore Hotel. The speaker of the evening was Dr. Hugh L. Dwyer (K. C. V. C. '11), formerly of Tulane University Medical School, who discussed the certified milk situation in the Kansas City territory. Dr. Dwyer is one of the leading pediatricians of Kansas City and his talk was replete with valuable information.

The program of the meeting on March 21 was devoted to chick problems. Dr. H. G. Beauman, of Drexel, Mo., spoke on the "Selection of Eggs, Incubation, Brooding and Care of Baby Chicks." Dr. Beauman runs a commercial hatchery in connection with his practice, and his talk was entirely authoritative. Dr. H. L. Bussong, of Belton, Mo., told of his experience in handling flocks of chicks affected with coccidiosis. Dr. S. J. Schilling, of the Jensen Salsbery Laboratories, discussed phases of pullorum disease control . The attendance at this meeting was 35.

Small-animal practice was discussed at the meeting on April 18. Dr. J. C. Flynn talked about rabies and rabies immunization, and the treatment of dogs bitten by rabid animals. Dr. J. M. Cozart, of Plattsburg, Mo., related some of his experiences in treating skin diseases of dogs. Dr. Arthur Trickett conducted a general round-table discussion on small-animal practice. Forty-seven persons attended this meeting.

The Association usually holds one monthly meeting during the spring at the laboratories of the Food and Dairy Division of the City Health Department. Fifty-eight members and visitors attended that meeting on May 16. The program was arranged to feature subjects pertaining to the city milk supply. Mr. Coe S. Pritchard, manager of the Pure Milk Producers' Association, Kansas City, gave an interesting talk on "Feeds and Feeding." Much interest was shown in the newer ideas

of feeding, especially for the production of special vitaminized brands of milk.

Mr. O. C. Murphy, director of the Food and Dairy Division of the City Health Department, discussed soft-curd milk and its relation to public health. Soft-curd milk is increasing in popularity and is especially recommended in some cases of infant and invalid feeding. An important part of the discussion was concerned with the wholesomeness of some soft-curd milk. Some cows normally give soft-curd milk. It was suggested that cows affected with certain types of udder infection may give soft-curd milk as long as they are so affected. Not all such cows, however, show clinical symptoms that may be picked out unless the strictest precautions are taken on making examinations of the udder.

A general discussion of current problems confronting the practitioners doing monthly and semi-annual herd inspections was carried on by Mr. Murphy. Both the veterinarians and health officials were enthusiastic about the meeting.

J. D. RAY, *Secretary.*

McLEAN COUNTY VETERINARY MEDICAL ASSOCIATION

The spring meeting of the McLean County Veterinary Medical Association was held in the pavilion at Miller Park, Bloomington, Ill., Sunday afternoon, April 30, 1933. About twenty-five veterinarians were in attendance from nine counties in central Illinois. A number of these had traveled over fifty miles to attend the meeting. Dr. A. B. Pletsch, of McNabb, Ill., presided.

Dr. W. H Welch, of Springfield, spoke at considerable length on the horse situation in Illinois. He said that the war had destroyed the man power as well as the horse power on the average American farm and that, although there were numerous indications that the farmer was returning to the use of horse power, this probably was only temporary. Dr. Welch pointed out that psychology played an important part in this question and that most farmers preferred to raise and sell grain rather than be bothered with the chores which are a necessary part of raising live stock. Dr. Welch deplored this state of affairs but he said that he believed he was presenting the situation in its true light. The discussion which followed brought out the fact that the ten million or more horses that have disappeared from

this country within the past twenty years would go a long way toward consuming the present grain surplus.

Dr. J. A. Owens, of El Paso, discussed the Besher plan for blending gasoline with alcohol made from corn. Dr. Owens spoke also of certain activities of the Illinois Agricultural Association with reference to the legislation then before the Congress in support of the Besher plan.

Dr. D. F. Luckey, secretary of the Illinois State Veterinary Medical Association, briefly reviewed the activities of his office in an effort to bring about correction of some of the abuses of the government in business. Dr. L. N. Morin, of McLean, Ill., followed Dr. Luckey and reviewed recent activities of the A. V. M. A. Special Committee on Agricultural Extension Service. Mr. Harry Heiser, public accountant, discussed in a very forceful way the activities of county agents and farm bureaus in Illinois, to show how these agencies had interfered with private enterprise of one kind or another.

Dr. H. Preston Hoskins, secretary of the American Veterinary Medical Association, briefly outlined plans for the 70th annual convention to be held in Chicago in August. This was the first time that Dr. Hoskins had been present at a meeting of the McLean County organization, and he acknowledged that he was of the opinion that reports of previous meetings of the Association had not been exaggerated. Announcements were made concerning the clinic of the Mississippi Valley Veterinary Association to be held at Peoria, on July 13, and the meeting of the Missouri State Veterinary Association to be held in Saint Louis, July 18-19.

By a unanimous vote, a resolution was adopted to the effect that Dr. Luckey should be retained as Executive Secretary of the Illinois State Veterinary Medical Association until after the adjournment of the present General Assembly.

J. S. KOEN, *Secretary.*

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Michigan-Ohio Veterinary Medical Association was held at the Courthouse, Adrian, Mich., May 11, 1933. The attendance was twenty-five, which is above the average for the past five years.

The program was rather unusual in that it consisted of discussions on a number of common medicinal agents used by vet-

erinarians in a general practice. The purpose of the program was to refresh the memories of the veterinarians in materia medica and therapeutics. Too many practitioners are allowing the manufacturing pharmacists to do their therapeutic thinking and prescribing.

Dr. A. Z. Nichols, of Hillsdale, Mich., opened the program with a talk on colocynth. Dr. J. W. Marshall, of Genoa, Ohio, followed with a paper on phytolacca. Dr. Lee Davisson, of Manchester, Mich., read a paper on sodium bicarbonate. Dr. C. F. Tuthill, of Onsted, Mich., was assigned iron sulphate and potassium dichromate. Dr. S. G. Colby, of Monroe, Mich., discussed the uses of magnesium sulphate. Dr. B. J. Killham, extension specialist from Michigan State College, East Lansing, described plans for a campaign for controlling bots in Michigan. He also discussed measures for controlling drugs in cattle. All of the subjects presented brought out more than the usual cussion, especially from veterinarians who frequently are silent on such occasions.

E. C. W. SCHUBEL, *Secretary.*

SIXTH ANNUAL CONFERENCE OF WORKERS IN PULLORUM DISEASE CONTROL

The sixth annual Conference of Workers in Pullorum Disease Control was held in the State Board of Agriculture Building, Dover, Del., May 15-17, 1933. Twenty-five members and ten outside speakers and invited guests were in attendance from the states of Alabama, Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Vermont, Virginia and West Virginia, the provinces of Ontario and Quebec, and the United States Department of Agriculture.

The program consisted of reports on the activities of the Antigen Committee; information concerning the pullorum disease eradication work of the different states and provinces, submitted by the various members and compiled by Dr. C. L. Martin; report of the Committee on Uniform Terminology; the preparation and use of pullorum disease stained antigen for the rapid, whole-blood test; description of an antigen nephelometer solidified with pectin; intermittency of reactions to the agglutination test; the agglutinability of some of the related organisms as applied to the doubtful reactor; discussion of the non-specific

reactor; presence of pullorum disease in pullets; the spread of pullorum disease through fecal contamination; short-interval testing; pleomorphism of the pullorum organism; and pullorum disease in turkeys.

The following poultry diseases also were considered: laryngotracheitis, coccidiosis, leukosis, fowl paralysis, paratyphoid in turkeys, mycosis in chicks, fowl-pox immunization and blackhead in turkeys.

Summary of 1932-33 testing data submitted by the laboratories.

Laboratory	Samples		Flocks
	Number Tested	Positive %	
Connecticut	180,755	0.8	244
Cornell	99,242	5.7	189
Delaware	90,004	3.9	319
Farmingdale	4,170	0.4	10
Maine	137,553	0.5	218
Maryland	87,701	22.0	369
Massachusetts	300,714	0.5	335
New Hampshire	237,219	0.5	209
Ohio	75,607	9.0	99
Ontario Agr. & Vet. College	173,296	3.0	543
Pennsylvania	366,787	4.8	715
Rhode Island	12,892	5.2	17
Vermont	28,372	2.6	56
Virginia	170,665	8.9	758
West Virginia	27,809	6.7	210
Totals	1,992,786		4,291

A representative from the U. S. Department of Agriculture presented a paper on the activities of the Food and Drug Administration in the control of misbranded poultry remedies.

Correspondence relative to the 1934 Conference should be addressed to Dr. J. S. Glover, Ontario Veterinary College, Guelph, Canada, at which place the 1934 Conference will meet.

Doctor Brown Enters Commercial Field

Dr. Frank H. Brown (Ind. '10), who was State Veterinarian of Indiana for seven years, has organized the Veterinary Bi-Ser-Pharm Supply House, at 3421 Kenwood Ave., Indianapolis, Ind., and will distribute Ashe Lockhart, Inc., biologics, National Laboratories anti-hog cholera serum and virus, and Curts-Folse pharmaceuticals, in Indiana and Ohio.

NECROLOGY

FREDERICK RANDOM WHIPPLE

Dr. Frederick R. Whipple, of Chicago, Ill., died at his home suddenly, May 28, 1933. Heart disease, from which he had suffered for several years, was the cause of death.

Born at Rutland, Ill., November 18, 1877, Dr. Whipple attended public schools and the McKillip Veterinary College. Following his graduation in 1902, he located at Kewanee, Ill. Several years later, he accepted the chair of Veterinary Science at

DR. F. R. WHIPPLE

West Virginia University, Morgantown, W. Va., where he remained for several years, then going to Sioux Falls, S. D., for a short time. He returned to Peoria and remained there until 1928, when he removed to Chicago and established the Broadway Animal Hospital.

Dr. Whipple joined the A. V. M. A. in 1925. He served as chairman of the Section on Small Animals, 1926-27. He was a member of the Illinois State Veterinary Medical Association and the Illinois State Board of Veterinary Examiners. He was

prominent in Masonic circles, was a past commander of the Peoria Commandery, Knights Templar; a member of Mohammed Temple of the Shrine and several other Masonic bodies. Funeral services were under Masonic auspices at Peoria and burial was at Springdale.

WALTER G. SARVER

Dr. Walter G. Sarver, of Tiskilwa, Ill., died on December 26, 1932, after a long illness.

Born near Scottsburg, Ind., January 26, 1879, Dr. Sarver attended local schools and then took up farming. Later he decided to study veterinary medicine. He entered the Indiana Veterinary College in 1914 and was graduated in 1917. The following year he entered the service of the U. S. Bureau of Animal Industry and was stationed in Richmond, Va., on tuberculosis eradication work. On October 1, 1918, he was transferred to Indianapolis on similar work, and three months later to Chicago on meat inspection. He resigned from the service early in 1919 and entered general practice in Tiskilwa, where he remained until 1927. He then was reinstated in the Bureau service and assigned to meat inspection at Pittsburgh. He again resigned on February 29, 1928, and entered practice at Princeton, Ill., where he remained until June, 1932, when he again located at Tiskilwa.

Dr. Sarver is survived by his widow, five daughters and one son.

WILLIAM J. MARTIN

Dr. W. J. Martin, prominent Illinois practitioner, passed away suddenly, May 27, 1933, from a heart attack, at Kankakee where he had spent the entire period of a long professional life.

Born in Kane County, Illinois, March 16, 1857, Dr. Martin was graduated from the Chicago Veterinary College with the class of 1897, after an attendance of one college session, having been admitted to advanced standing from the Ontario Veterinary College. The deceased belonged to the pioneer rural veterinarians of Illinois, having entered into practice at Kankakee at a time when few veterinarians had attempted to develop a clientele in a rural community. Besides being a prominent figure in the development of the Illinois State Veterinary Medical Association, which he served as president in the early part of

this century, Dr. Martin devoted much time and pains in writing contributions on practical subjects for the contemporary literature of the forming profession. He was a collaborator on the editorial staff of the American Veterinary Review from 1899 until 1901. Due to a forceful personality, sound judgment, honesty of purpose and untiring efforts, Dr. Martin won and retained to the end an outstanding position of respect and admiration among his colleagues.

Dr. Martin joined the A. V. M. A. in 1899 and served as chairman of the Committee on Pharmacy (1899-1902) and as a member of the Audit Committee (1918-20 and 1922-23). For a number of years Dr. Martin was a member of the Illinois State Board of Veterinary Examiners. He is mourned by a daughter and two sons, one of whom, his successor, is Dr. D. C. Martin (Chi. '16), who was a major in the veterinary service of the American Expeditionary Forces during the late war.

L. A. M.

RALPH MACK BELL

Dr. Ralph M. Bell died suddenly at his home in Riverside, Calif., on May 24, 1933, of cardiac failure. He had been in apparently perfect health up to the minute of his death.

Born in Ceres, N. Y., June 8, 1877, Dr. Bell attended Alfred University. He later entered the New York State Veterinary College at Cornell University and was graduated in 1909. He engaged in general practice in New York State until 1913, when he went to the University of California, where he was engaged in research work on tuberculosis for three years. He was joint author of several articles covering these studies.

In 1915, Dr. Bell was employed as veterinarian for the Miller & Lux Company, one of the largest land and cattle owners in the United States. The same year he returned to New York State to engage in practice, and a short time later entered the Veterinary Corps of the U. S. Army and served during the war with the rank of captain. In 1925, he returned to the University of California, where he remained until 1927, when he resigned to enter private practice at Riverside. He was also City Veterinarian of Pasadena.

Dr. Bell joined the A. V. M. A. in 1929. He was a member of the California State Veterinary Medical Association and the Southern California Veterinary Medical Association. He is survived by his widow (née Edna G. Babcock). Burial was at Riverside.

ELWIN J. CREELY

Dr. Elwin J. Creely, of San Francisco, Calif., committed suicide, May 17, 1933, by inhaling fumes from his automobile exhaust. He was born in San Francisco, April 20, 1892, and was a graduate of the San Francisco Veterinary College, class of 1914. Shortly following his graduation, Dr. Creely entered the service of the U. S. Bureau of Animal Industry and served at various stations, including Chicago, San Francisco, Reno, Salt Lake City, Portland and Sacramento.

During the late war, Dr. Creely was on furlough from the B. A. I. service, from June 22, 1918, until August 6, 1919. He served as a second lieutenant in the Veterinary Corps. Dr. Creely terminated his B. A. I. service on December 31, 1927.

Dr. Creely joined the A. V. M. A. in 1920. He is survived by his mother, two sisters and two brothers, one of whom is Dr. A. J. Creely (San Fran. '15), of Boise, Idaho. The deceased was the son of the late Dr. Edward J. Creely, founder of the San Francisco Veterinary College, and the nephew of Dr. T. R. Creely (San Fran. '07).

MALCOLM JAMES HARKINS

Dr. Malcolm J. Harkins, Conshohocken, Pa., died June 4, 1933, at Temple University Hospital, Philadelphia, at the age of 41. His death occurred quite unexpectedly. It was due to purulent nephritis and followed an infected finger from which he had suffered for several weeks before being taken seriously ill.

During his active career, which was so untimely terminated, Dr. Harkins was engaged in many phases of scientific work. Immediately following his graduation from the University of Pennsylvania, in 1912, he began work with H. K. Mulford Company, where he continued for several years. During a part of this time he was a member of the veterinary faculty of his Alma Mater. Later on, Dr. Harkins became affiliated with the Research Institute for Cutaneous Diseases, in Philadelphia, and finally changed over to general practice in his home town, in which work he was engaged at the time of his death.

Dr. Harkins joined the A. V. M. A. in 1913 and served as secretary of the Section on Education and Research, 1927-28. At the time of his death, he was a member of the Committee on Veterinary Biological Products, having served one year (1928-29) as chairman. He was a member of the Keystone Veterinary

Medical Association and the Pennsylvania State Veterinary Association, and was respected by all as a progressive veterinary scientist. He is survived by his widow and five young children, for whom we all feel the greatest sympathy.

A. H. C.

JAMES T. ROSS

Dr. James T. Ross, one of the oldest practitioners in the Philadelphia area, died at his home in Frankford, Philadelphia, June 6, 1933, in his 73rd year. Next March he would have completed his fiftieth year in practice. Except for the infirmities of age, he was in fair health until a few days before he died.

Dr. Ross entered the veterinary profession by the method in general use before veterinary schools were established, studying and working for four years with M. W. Birch, M.R.C.V.S., who had an extensive practice. Dr. Ross received his certificate of proficiency, March 11, 1884, and at once began practicing in Frankford, where he was born and reared. He continued there until his death.

When the University of Pennsylvania opened its veterinary school, in the fall of 1884, Dr. Ross matriculated as a student, but his efforts to conduct a practice and attend classes at the same time affected his health and he was obliged to withdraw. He continued to study, however, and having a retentive mind and good judgment, he made good use of his library, which he kept up to date. He had been a subscriber to the JOURNAL for many years.

Dr. Ross had a large practice in his most active years and was widely known in his community as an able veterinarian and a man of sterling character. He is survived by his widow, two sons and two sisters.

A. H. C.

JAMES A. McGAVICK

Dr. James A. McGavick, of Tacoma, Wash., was drowned in a lake near Mount Rainier while fishing on June 18, 1932.

Born at Rosedale, Wash., August 23, 1902, Dr. McGavick attended grade and high schools and then entered the State College of Washington. He was graduated with the degrees, B. S. and D. V. M., in 1924, and for a time was employed as a state veterinary inspector. At the time of his death, Dr. McGavick was City Veterinarian of Tacoma.

Dr. McGavick joined the A. V. M. A. in 1931. He was a member of the Washington State Veterinary Medical Association and Alpha Psi Fraternity.

He left a widow (née Leah De Rochier), one son, two brothers, one sister and his parents. Dr. McGavick's mother has died since his death. R. A. B.

EARL MAX PICKENS

Dr. E. M. Pickens, Professor of Bacteriology and Pathology at the University of Maryland, College Park, died very suddenly while in conference at the University, June 13, 1933. Death evidently was due to heart failure.

DR. E. M. PICKENS

Born in Parish, N. Y., January 29, 1888, Dr. Pickens studied veterinary medicine at Cornell University. He received his D. V. M. in 1911 and two years later his Master of Arts, also from Cornell. He remained at Ithaca as Instructor, then Assistant Professor and later as Professor of Bacteriology and Pathology, under the late Dr. Veranus A. Moore. In 1918, Dr. Pickens was called to the University of Maryland to take the position of Professor of Bacteriology and Pathology, which he held at the time of his death. He was also in charge of the Biological and Live Stock Sanitary Service Laboratories at the University of Maryland. For many years he was a member of the editorial staff of the *Cornell Veterinarian.*

Dr. Pickens joined the A. V. M. A. in 1915 and rarely missed attending an annual convention. He was the first secretary (1922-26) and later chairman (1926-27) of the Section on Education and Research, and did much to establish the high standards maintained by the programs of the Section. Dr. Pickens served also as A. V. M. A. Resident Secretary for Maryland from 1927 until the time of his death. The A. V. M. A. membership in Maryland showed a very substantial growth during this period.

For over nine years, Dr. Pickens had been Secretary-Treasurer of the Maryland State Veterinary Medical Association, and the efforts he had put forth were largely responsible for the healthy condition of the organization during recent years. Dr. Pickens was a Fellow of the American Public Health Association and, at the time of his death, was secretary of the Section on Food and Nutrition in that organization. Dr. Pickens held membership also in the Society of American Bacteriologists, the American Genetics Association and the U. S. Live Stock Sanitary Association. He was a member of Omega Tau Sigma, Phi Delta Theta, Gamma Alpha and Sigma Xi fraternities.

The funeral, conducted under Masonic auspices, was one of the largest ever held in College Park. Members of the veterinary faculty at Cornell University, practically the entire membership of the Maryland State Veterinary Medical Association and a large number of veterinarians from the U. S. Bureau of Animal Industry were present. Dr. Pickens was buried in Fort Lincoln Cemetery in the District of Columbia.

Surviving Dr. Pickens are his widow, one son and two daughters.

JOHN PERSCHBACHER

Dr. John Perschbacher, Janesville, Wis., died in Mercy Hospital, Janesville, June 17, 1933, the result of a fractured skull and other injuries received in an automobile accident six miles east of Janesville four days previously.

Born in the Province of Ontario, Canada, January 24, 1861, Dr. Perschbacher studied veterinary medicine at the Ontario Veterinary College. Following his graduation in 1901, he located in Janesville and practiced there continuously until his death.

Dr. Perschbacher joined the A. V. M. A. in 1928. He was a member of the Wisconsin Veterinary Medical Association and of Western Star Lodge No. 14, F. and A. M., of Janesville. He

is survived by his widow (née Anna Wiegand), one brother and two sisters.

CYRUS P. DRAPER

Dr. C. P. Draper, of Arlington Heights, Ill., died at his home, June 17, 1933, after an illness of about a year. He was a graduate of the McKillip Veterinary College, class of 1902, and had been engaged in tuberculosis eradication in McHenry County for about ten years. Dr. Draper is survived by his widow and one daughter.

ORLAND J. MOON

Dr. Orland J. Moon, of New Holland, Ohio, died suddenly at Bear Lake, near Battle Creek, Mich., June 16, 1933, a victim of an apoplectic stroke. His health had been impaired for some time.

Born May 26, 1881, he was reared in Sabina, Ohio, and attended the local grade and high schools before entering Ohio State University. Following his graduation from the College of Veterinary Medicine, in 1909, he located at New Holland and built up a fine practice in that territory. He had a number of business interests, including the Moon Oil Company, which he owned and managed. Just recently he had completed a five-year term as trustee of the Pickaway County Children's Home.

Dr. Moon is survived by his widow (née Lela Sapp), his mother, two sisters and two brothers.

CHARLES F. NORD

Dr. Charles F. Nord, Omaha, Neb., died suddenly of heart failure June 20, 1933, shortly after he started work in one of the packing-plants in South Omaha. He had been in apparent good health up to the time of his death.

Born in Denison, Iowa, April 23, 1876, Dr. Nord attended local grade and high schools and Denison Academy. He studied veterinary medicine at Kansas City Veterinary College and, following his graduation in 1912, he returned to Denison for practice. He served in the Veterinary Corps during the late War and for a time was stationed at Fort Sam Houston, Texas. In April, 1927, he was appointed veterinary inspector by the U. S. Bureau of Animal Industry and was assigned to meat inspection in Omaha.

Dr. Nord joined the A. V. M. A. in 1918. He was a member of the National Association of B. A. I. Veterinarians. He was a Mason and a Shriner. He is survived by his widow (née Amanda E. Conquist), one son and two brothers. Burial was at Kiron, Iowa.

Come to Chicago—August 14-15-16-17-18

PERSONALS

MARRIAGES

Dr. Mark W. Allam (U. P. '32) to Miss Lyla J. Griswold, both of Media, Pa., April 15, 1933, at Media, Pa.

Dr. G. Thatcher Parker (Corn. '32), of Red Bank, N. J., to Miss Ruth Cross, April 15, 1933, at Cleveland, Ohio.

BIRTH

To Dr. and Mrs. R. A. Hendershott, of Trenton, N. J., a son, John Abbott, December 31, 1932.

PERSONALS

Dr. O. F. Cox (Colo. '21) has removed from Clarion, Iowa, to Defiance, same state.

Dr. Cliff D. Carpenter (Corn. '20) has removed from Van Nuys, Calif., to Los Angeles, Calif.

Dr. D. A. Eastman (Iowa '22), of Moline, Ill., has disposed of his hospital in Cedar Rapids, Iowa.

Dr. John L. Tyler (Chi. '89) has changed locations from Oroville, Calif., to North Hollywood, Calif.

Dr. J. H. Howard (Corn. '32) is associated with Dr. M. C. Markham (Corn. '18), of Binghamton. N. Y.

Dr. Daniel P. Ehlers (K. S. C. '28) reports a change of address from Harrisburg, Pa., to Butler, Pa.

Dr. R. A. Gregory (K. C. V. C. '09) now receives his mail at Gaithersburg, Md., instead of Washington Grove, Md.

Dr. H. M. Aitken (O. S. U. '31), formerly of Columbus, Ohio, is now located at 500 East High St., Springfield, Ohio.

Dr. L. F. Maier (O. S. U. '15) has requested a change of address from Apponaug, R. I., to North Scituate, same state.

Dr. Maurice C. Hall (Geo. Wash. '16), of Washington, D. C., has been a hospital patient for about seven weeks.

Dr. E. L. Cornman (U. P. '00), of Marietta, Pa., has been elected president of the Exchange National Bank of Marietta.

Dr. J. C. Wickham (O. S. U. '12), formerly of Wooster, Ohio, is now in Cleveland, Ohio. His address is: 2337 Broadview Road.

DR. HENRY VAN ROEKEL (Iowa '25) left New Haven, Conn., on June 1, to return to Massachusetts State College, Amherst, Mass.

DR. A. E. HANCOCK (O. S. U. '31) is operating a modern hospital for small animals at 122 East Main St., Lancaster, Ohio.

DR. E. M. KEEF (San Fran. '13), formerly of Oakland, Calif., has removed to San Mateo, Calif. Address: 920 South Eldorado St.

DR. R. S. MONTGOMERY (K. C. V. C. '17), of Highland Park, Ill., was elected to membership in the Highland Park Lions Club in May.

DR. EDWIN C. F. ENGE (U. P. '31), formerly of Drexel Hill, Pa., gives a new address: Lansdowne Avenue near State Road, Upper Darby, Pa.

DR. G. A. JONES (K. C. V. C. '12), of Sedro-Woolley, Wash., is now serving as mayor of Sedro-Woolley. He was elected the first of the year.

DR. S. K. HAZLET (Chi. '95), of Oelwein, Iowa, is reported to be suffering from an illness which may prevent him from practicing again.

DR. GEORGE GORDON (Chi. '14), of Chicago, Ill., was the speaker of the evening at the monthly meeting of the Chicago Persian Society on May 20.

DR. WALTER W. WILLIAMS (Corn. '15), of Springfield, Mass., was graduated from the Albany Medical College, Union University, on June 12.

DR. GEORGE H. KINEMOND (U. P. '26) announces the removal of his offices from 1844 North Park Avenue to 6448 Germantown Avenue, Philadelphia, Pa.

DR. W. S. PLASKETT (McGill '93), of St. Petersburg, Fla., will spend the summer months at Osoyoos, British Columbia. He will then return to St. Petersburg.

DR. FRANK J. LINGO (Cin. '08), of Albuquerque, N. M., was injured in an automobile accident on March 18 and was just beginning to get around early in June.

DR. THOMAS F. COLLING (Ont. '02), formerly of Burnaby, British Columbia, is now located at Newton in the same province, where he is operating a small farm.

R. A. RUNNELLS (Mich. '16), Associate Professor of Veterinary Pathology at Iowa State College, is doing graduate work at the University of Michigan again this summer.

DR. CECIL MOULTON (San Fran. '18), formerly of San Rafael, Calif., is now stationed at San Leandro, Calif., on meat inspection for the California Department of Agriculture.

DR. H. T. CLARNO (Chi. '18), of Farmer City, Ill., has located at LeRoy, Ill., for general practice, after having been in the employ of the Illinois State Department of Agriculture for about six years.

DR. JOHN E. MCCOY (K. S. C. '09), who has held the position of Professor of Veterinary Medicine at the State College of Washington for the past ten years, has resigned to give his attention to private business.

DR. C. J. MARSHALL (U. P. '94), of the University of Pennsylvania, addressed the Northwestern Pennsylvania Veterinary Medical Club at Sharon, Pa., on April 18, on the subject, "Sweet Clover Poisoning in Cattle."

DR. JAMES F. MITCHELL (Corn. '11), who has been with the Cerro de Pasco Copper Corporation in South America for a number of years, reports a change of address from Oroya to Pachacayo, Peru, c/o Hacienda Pachacayo.

DR. A. C. SCHAFSTALL (Ont. '10), of New Washington, Ohio, landed in the ditch when his car collided with another on the road near Lykens, Ohio, recently. Dr. Schafstall escaped unhurt and his car was only slightly damaged.

DR. A. E. CAMERON (Ont. '08), Chief Veterinary Inspector, Health of Animals Branch, Canada Department of Agriculture, sailed for Great Britain, on June 30, to be abroad for two months.

DR. G. W. JENSEN (Chi. '18), of Antioch, Ill., was seriously injured in an automobile accident near Long Lake on May 18. Dr. Jensen sustained fractures of the pelvis and five ribs in addition to other injuries of the head and limbs.

DR. H. A. TRIPPEER (McK. '07), who has been in the sheep-raising business, has returned to his old location at Walla Walla, Wash., where he has been appointed to the full-time position of City Milk Inspector and Deputy County Health Officer.

DR. E. L. STUBBS (U. P. '11), professor of veterinary pathology at the University of Pennsylvania, addressed the Schuylkill Valley Veterinary Medical Association at the meeting held in Hamburg, Pa., on April 20. His subject was "Diseases of Poultry."

DR. L. T. KILFOY (K. C. V. C. '16) has opened a new hospital for small animals at 1529 Lawrence Ave., Chicago, Ill. Dr. Kilfoy's old hospital, at 2542 Lawrence Avenue, was badly wrecked by an explosion in a near-by cleaning plant several months ago.

DR. WILLIAM PISTOR (Wash. '26), who has been employed by the University of California in research work at Petaluma, Cal., has accepted appointment as Professor of Veterinary Medicine at the State College of Washington, and will report for duty, September 16.

DR. D. H. EASTMAN (O. S. U. '22), of Aledo, Ill., has resigned as assistant state veterinarian, after ten years spent in tuberculosis eradication work, and will engage in general practice. Dr. Eastman is secretary of the Mercer County (Ill.) Veterinary Association.

DR. HIRAM T. GAETZ (U. P. '03), of Buffalo, N. Y., attended the meeting of the alumni of the Veterinary School of the University of Pennsylvania at Philadelphia, Pa., on May 20, and received the prize awarded to the alumnus who had traveled the greatest distance for the express purpose of attending the meeting.

DR. E. A. RODIER (Wash. '20), for the past six years veterinary pathologist of the Veterinary Research Division of the Bureau of Animal Industry, Phillippine Department of Agriculture, has resigned to accept a position as director of the Bureau of Animal Disease Control under the Department of Reconstruction for the province of Kwangsi, South China.

DR. W. R. CRAWFORD (Corn. '24), of Westminster, Md., is able to look after his work again, following a disastrous automobile accident about two months ago. Dr. and Mrs. Crawford, with their infant daughter, were on their way to visit friends when the accident occurred. Another car crashed into their car and pinned it against a telephone pole. The sympathies of their many friends, both in and out of the veterinary profession, are with Dr. and Mrs. Crawford in the death of the daughter who died as a result of the accident. Mrs. Crawford is slowly recovering from her injuries.

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